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GENESIS OF CEREBELLAR FUNCTIONS

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In this discussion of the cerebellum, an attempt is made to visualize the genesis of cerebellar functions.

Realizing how far in the future definitive conclusions in this subject must be, I prefer to consider this communication as a study embodying certain of my present views, leaving my position still flexible and open to whatever modifications further investigations may dictate.

Many difficulties have previously been encountered in reaching a solution concerning cerebellar functions. Indeed, it seems almost impossible to draw any comprehensive deductions regarding this question from all of the evidence at present available. Animal experimentation and clinicopathologic observation in mammals and man leave much to be desired. A vast amount of experimental work has been carried on without the necessary preceding morphologic understanding of the cerebellum, and the results, in consequence, have often confused rather than clarified the situation. By far the majority of workers in the past have directed their attention to the organ at a stage in its development when it has reached its highest complexity. This necessarily has imposed all of the difficulties of approaching the problem in its most complex rather than in its simplest form.

It is my intention to pick up the threads in the genesis of this organ where they may be traced separately in the most primitive forms of vertebrate life, before they have woven themselves inextricably into the complex fabric of the ultimate cerebellum. Starting from this, its organic inception, it may be easier to follow step by step the progressive evolution of structure which has gone hand in hand with the advancing development of motor control as the vertebrates acquired new capacities and adjustments.

The citation of authorities on this subject is a matter so familiar that it seems unnecessary to include here the various opinions formerly expressed. The reader, however, is especially referred to the works of André-Thomas, Babinski and Tournay, Luciani, Bechterew, Bârâny, Bolk, Van Rynberg, Mills and Weisenburg.

I wish to offer a word of apology in connection with the nomenclature. In order to place the cerebellum in proper alinement for the description of certain evolutional phases in its genesis, it has seemed necessary to select for it the term parencephalon. This brings the terminology into more precise consonance with that of the other brain vesicles, such as diencephalon and mesencephalon. Although the addition of another term to an already overburdened vocabulary is perhaps unfortunate, this one is selected advisedly, because it was employed by Aristotle, who called the cerebellum "parencephalis."

The method of approach in this investigation includes comparative morphologic studies beginning with the more primitive forms of vertebrate organization, such as cyclostomes, sharks and rays, ganoid and teleost fish; with later reference to reptiles, amphibians, birds and mammals. The embryogenetic characters of the cerebellum were studied in all of these forms with the idea of revealing a common ground-plan in the structure of the organ, together with an analysis of the motor reactions of the animal in the light of its cerebellar organization. Some preliminary experimental work was done on sharks, pigeons and cats.

The problem of this research may be phrased in three queries: 1. When, in the course of animal organization, did the cerebellum make its first appearance? 2. What were the probable morphologic and physiologic conditions which established the demand for this organ, and what evidence is there that the cerebellum in its primordial form became essential to the posture-maintaining quality of motor control? 3. In what manner did the cerebellum, once having acquired its specialization for dominating the posture-maintaining elements of motor control, eventually establish within itself a hierarchy regulating all of the further expansions and modifications of this quality of motor activity?

THE FIRST APPEARANCE OF THE CEREBELLUM

The first query may be answered categorically in the statement that no exact homologue of the vertebrate cerebellum is known in the invertebrate phylum. The cerebellum, therefore, made its appearance at some critical period during the invertebrovertebrate transition in response to certain functional necessities which did not exist in the invertebrate.

It might appear unwarranted, however, to assume so dogmatic a position without first considering some of the conditions of invertebrate life. Insects, for example, must utilize some stabilizing and balancing mechanism for the delicate adjustments of their bodies in flight through the air, in alighting or in starting to fly. Furthermore, such invertebrates as move about in the water, employing various means of locomotion, must likewise be in need of a definite equilibratory apparatus, and this also applies, perhaps in a less degree, to land-living insects or other invertebrates which crawl or creep.

In the king crab (*Limulus polyphemus*), Patten [1] found by experiment that there is a part of the central nervous system the destruction of which produces a marked disturbance in the animal's equilibrium. After cutting or removing the vagal neuromeres, it is impossible for the animal to right itself if placed on its back in the water. If the experiment is confined to one side, the crab constantly moves in a circle to the side of the operation. The circular movements continue for hours at a time. Incision or extirpation involving any part of the brain behind or in front of these vagal neuromeres does not destroy the tendency or power of the animal to right itself or properly direct its locomotion when able to move.

Patten [1] is of the opinion that the subdivision of the incipient vertebrate head is to be observed in the arachnids, and that the brain vesicles follow a plan of subdivision analogous to that in vertebrates. The fourth brain vesicle in arachnids is the metencephalon or vagus organ. The lateral stomodeal ganglion is connected with this part of the brain and is united by a large commissure forming a prominent arch over the neural surface of the stomodeum. It is gradually crowded caudad by the migration of the mouth and rostrum, as well as by the increasing size of the lateral eyes, forming in vertebrates the rudiment of the cerebellum.

Although Patten [1] believes that the median stomodeal ganglion of arthropods and the cerebellum of vertebrates are the only brain structures that arise originally in the median line, it is by no means clear that this median stomodeal ganglion is in any sense the homologue of the vertebrate parencephalon. Indeed, there is much doubt in the light of the embryogenetic process which results in the formation of the cerebellum in the lowest vertebrates, whether this median stomodeal ganglion is in any sense homologous with the parencephalon. The mode of development of the cerebellum in the cyclostomes and in all the true fish points more conclusively to a bilateral paired anlage than to a median unpaired mode of derivation.

So for the present at least, and until much more extensive studies in vertebro-invertebrate homology may be completed, it is fair to assume that the vertebrate cerebellum has no exact counterpart in the invertebrate phylum, and that this organ should be regarded as a distinctive vertebrate character. It came into existence during an epoch-making period when a critical transition led to the vertebrate differentiation out of some invertebrate line. Thereafter it has maintained itself with increasing prominence as one of the chief features in the vertebrate brain. Even thus interpreted, it must still remain a question whether

1. Patten, W.: The Evolution of the Vertebrates and Their Kin, Philadelphia, 1912.

the cerebellum is a neomorph. It is, perhaps, more far-seeing to regard its fundamentals as already laid down in some diffuse invertebrate rudiments ultimately assembled as a distinctive vertebrate organ under the influence which determined the inception of the vertebrate phylum.

THE MORPHOLOGIC AND PHYSIOLOGIC CONDITIONS LEADING TO THE DEVELOPMENT OF THE CEREBELLUM

Consideration of the next phase of the problem deals with the probable morphologic and physiologic conditions which established the demand for this organ. This opens directly into one of the most fertile fields of evolutional research, namely, the mode of transition from the invertebrate to the vertebrate.

The evidence furnished by living and fossil forms is by no means sufficient to be entirely convincing concerning the nature of this process. In consequence, a number of theories have been advanced and have had earnest advocates, so that the subject is still a matter for debate. But whatever divergence of opinion may exist with reference to this

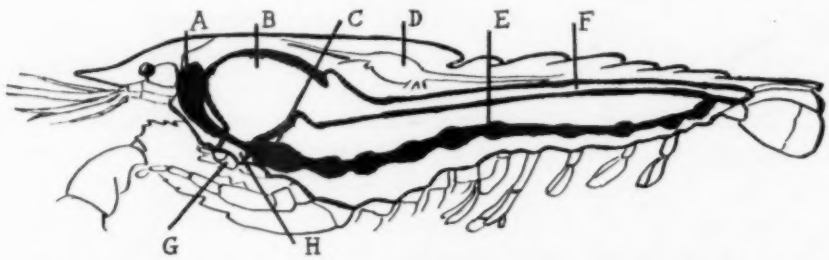

Fig. 1.—Midsagittal section of lobster (*Homarus americanus*) showing relation of the stomach and nervous system. *A* indicates supra-esophageal ganglion; *B*, stomach; *C*, infra-esophageal ganglion; *D*, heart; *E*, ganglionated cord; *F*, intestine; *G*, mouth; *H*, esophagus.

evolutional process, there is real unanimity concerning the most outstanding difference between the organization of the higher segmented invertebrate and that of the vertebrate. This difference depends on the position of the mouth, together with the relation of the alimentary canal to the nervous system, which relation the position of the mouth appears to determine (Fig. 1).

In the higher segmented invertebrates (arthropods, such as the king crab, lobster or cray-fish) the mouth occupies a position ventral to the lateral eyes and is continuous with a slender tube, the esophagus. This tube passes between the head ganglions to the stomach which lies as a dilated pouch dorsal to the brain. To all intents and purposes, therefore, the esophagus passes through the center of the brain, being surrounded on all sides by central nervous tissue.

The caudal prolongation of the nervous system extends toward the tail from the infra-esophageal ganglion, and lies ventral to the intestinal

canal. By this arrangement the entire nervous system occupies a position in front of the alimentary canal, which relation is exactly the reverse of the enteroneural relation in the vertebrates. Although this relation of the stomach and intestine to the central nervous system and the general configuration of the body may have certain mechanical advantages in aiding the animal to maintain the position best suited to its purposes of life, it is subject to one serious disadvantage. The esophagus being surrounded by the brain, at once becomes jeopardized as the conducting tube from the mouth to the stomach should the brain for any reason take on increased growth. By such growth in the brain the esophagus would become more and more constricted until reduced to the smallest dimensions, as it is in many insects which are for this reason forced to depend on fluid alimentation sucked through the narrow tubular esophagus into the stomach. Any evolutional development in the brain, therefore, by reducing the caliber of the esophagus, would threaten the whole process of food ingestion, and thus have profound effects on the metabolism of the animal.

This compromising relation of the esophagus to the brain which occurs in the invertebrate is completely changed in the vertebrate, for in such forms of animal life the stomach and intestine have assumed a position ventral to the nervous system, and the esophagus is no longer embarrassed by the necessity of passing through the brain in order to reach the stomach. Such a great and far-reaching alteration as this in the neuro-enteric relation has provided the brain with an opportunity to expand as it may without jeopardizing the alimentary canal or exacting a limitation in the food supply of the animal.

The perplexing question is: What were the influences and processes which produced this alteration in the relation of the nervous system to the digestive tube?

Geoffrey St. Hilaire[2] advanced the explanation that this great change was wrought by the simple process of the invertebrate turning over on its back and swimming in this position, thus gradually converting its dorsal into its ventral surface and causing the appearance of a new mouth, which opened in this case on the new ventral surface directly in the alimentary canal without passing into the brain, while the old mouth in the dorsal position became obliterated.

Another view concerning this epoch-making alteration of relations was advanced by Bateson,[3] who maintained that from the beginning there were two types of bilateral elongated segmented animals which differentiated along two distinctly different lines. In one the alimentary

2. St. Hilaire, G.: Sur la vertèbre, La rev. encyclopedique, 1822.

3. Bateson: The Ancestry of the Chordata, Quart. J. Micr. Sc., 1886, vol. 26. Materials for the Study of Variation, London, 1894.

canal was dorsal to the nervous system, and these animals in their highest evolutional form became the arthropods; while in the second, the nervous system was dorsal to the digestive tube from the beginning and reached the final evolutional stage in the vertebrates. The latter view, interesting as it may be, lacks the necessary evidence to establish these two distinctly different types of ancestral animal life, either living or fossil.

Among the most suggestive theories explaining the manner in which the nervous system assumed a relation dorsal to the gut tract in the vertebrate is the one proposed by Gaskell.[4] He recognized the necessity imposed by the invertebrovertebrate transition for a rearrangement of the relations in the neuraxis and the digestive tract, and he believes that this took place by a progressive invasion of the walls of the stomach by nerve cells lying in the brain ganglions in the invertebrate. Little by little this invasion of the pouch by neural elements converted the walls of the stomach into a solid mass of nerve tissue on either side, surrounding the old epithelial lined cavity of the stomach, which now becomes the ventricle of the brain. This invasion of the stomach is not complete, for in the region of its roof it still retains its old epithelial character even where the invasion of the nerve cells attains its most extensive representation in the brain of the higher mammals. Here, in this region of the roof, the epithelial lining of the old stomach is thrown into many glandular reduplications represented by actual glandular tissues in the different orders of vertebrates, thus showing the potentiality of the roof of the brain to manifest its old gastric proclivities in the formation of glandular structures. These glandular structures include the paraphysis and the dorsal sac, the choroid plexuses and the pineal gland.

After the invasion of the stomach by these nerve cells coming from the ventral brain ganglions of the invertebrate, the nerve cells from the ganglionated cord follow suit, invading the floor and side walls of the intestinal canal, thus forming the spinal cord whose central canal in the vertebrate represents the primitive invertebrate channel of the gut.

Gaskell [4] finds substantiation for this view in many facts related to the central nervous system, but the one of cardinal importance to him is the position of the infundibulum which in the embryo passes from the region of the mouth cavity into the third ventricle. This tube, lined with epithelium, is a remnant of the old invertebrate esophagus, and the fact that it has become invested by nerve tissue is simply another illustration of the neural invasion from the head ganglions. According to this view of Gaskell, it is not necessary for the animal to turn over and swim on its back. All it has to do is to

4. Gaskell, W. H.: The Origin of Vertebrates, London, 1908.

sacrifice a perfectly good stomach in the interests of forming a better brain, and also to give up an efficient gut tract in order that it may win a spinal cord.

This supposed critical exchange between brain and stomach probably points to the most glaring defect of Gaskell's theory, for if the invertebrate thus transformed its gut tract into a central nervous system, what was left to it for the purposes of alimentation? It may be in the line of efficient evolutional progress to produce a more highly developed nervous system, but a brain without a stomach to sustain it would bear little of promise to the animal.

Patten,[1] although in accord with Gaskell in certain main lines of the process which resulted in the changed neuro-enteric relation, differs materially with reference to the manner of this change. He, like Gaskell, believes that the line of ascent was through the arthropods and thence carried on by the ostracoderms, a class of animal midway between the most primitive vertebrates and the highest segmented invertebrates. These animals are now entirely extinct and seem to have been present only from the Silurian to the upper Devonian age.

Patten's interpretation of this transition draws on the imagination much less than does that of Gaskell. He recognizes the necessity of providing a new entrance into the gut tract in order to escape the constricting influence of brain growth on the esophagus. This, he believes, is accomplished by a gradual shifting of the old mouth from the ventral surface of the head into what, for the invertebrate, would be a dorsal position. As this gradual shifting took place, a mouth was established which provided a direct connection between the esophagus and the stomach, without passing through the brain. According to Patten's view, this relatively simple shifting of the mouth would then convert the ventral invertebrate surface into the dorsal vertebrate aspect of the body and determine a gut tract ventral to the central nervous system. The opportunity for developing such a new mouth was provided, according to Patten, by the presence of a very ancient organ in the arthropod head, known as the cephalic navel or dorsal organ. This stood ready to take the place of the old mouth which was slowly being eliminated. "Its presence alone made the existence of the vertebrates, as we know them, a possibility."

The development of a vertebral endoskeleton, according to most authorities, does not seem nearly so difficult to explain as this profoundly important relation of the nervous system to the digestive tube. When it was finally determined with the definitive arrival of the vertebrate, by whatever evolutional process it may be, animal organization had come into possession of an entirely new physical proposition. The stomach and intestine, which in the invertebrate occupied a position in

the back, in the vertebrate now occupied a position in the belly. Mechanically, this new relation of the central axis of the vertebrate to the gut and stomach imposed the necessity for new functions in maintenance of the best posture in which the animal might live. The intestine and stomach, filled as they were with the gaseous contents of the alimentary canal, made the ventral aspect of the body lighter, and unless some means were devised to hold the belly down, the animal would be turned over on its back and thus be deprived of its best opportunities for dealing with the elements of its environment. In order, therefore, to maintain what may be called the optimum physiologic posture, there arose the necessity for an organ which would so synergize the ventral, dorsal and lateral musculature as to hold this position.

The earliest vertebrates of which there is evidence, namely, the cyclostomes, comprise a group of rather sluggish water-living animals. The best known representatives of this group are the lampreys and the hagfish. These animals present all of the characteristic vertebrate features. They have a well demarcated head and a long segmented body, gills and a gut tract, which lie ventral to the brain and spinal cord. The mouth is large and circular, without jaws, and equipped with rough, small, stonelike teeth. These animals have a well marked dorsal fin and an inconspicuous ventral fin, but no paired lateral fins or other body appendages. In another important respect they differ from the invertebrates, i. e., in the possession of a series of organs situated along the lateral aspect of the body, on either side, connected with the brain, known as the lateral line organs, which function in the acts of balancing. In addition to these organs of balance, the cyclostomes have two semicircular canals in the head which are exquisitely sensitive to changes in position and are important structures in the maintenance of equilibrium.

Invertebrates appear to have no organs homologous to the semicircular canals. Some insects have specialized auditory organs which probably are more closely related to the sense of hearing than to the function of balance. Certain hairs scattered over the body of some insects and on the antennae are sensitive to sound and may be related to the sense of balance. The basal joint of the antennae of some insects forms a socket richly supplied with sense organs containing a large ganglion supposed to have a functional responsibility related to equilibrium. Nothing comparable to the semicircular canals, however, which made their first appearance in the cyclostomes, has been observed in invertebrates.

The life history of one of these primitive vertebrates sheds much light on the mechanical handicap imposed by the now ventrally placed alimentary canal (Fig. 2). Selecting as an example the lamprey (*Petromyzon*), it is found that after leaving the egg the young must

shift for themselves. At this period the animal is known as the ammocoete, or larval form of *Petromyzon*. For the first three years of its life, the ammocoete lies submerged in the mud in shallow waters. During this time it depends for its nutrition on what is brought to it by the moving currents of water above it. It lies flat on its belly with its dorsal fin directed upward. It has little independent motor life, except those slight activities necessary to maintain an optimum physiologic position. Its mouth is directed forward, while the position of the body is best calculated to facilitate its bilateral symmetrical growth. At this stage the nervous organization is relatively simple, although it contains all of the fundamentals of the ultimate vertebrate neuraxis. Its cerebellum consists of a heavy ridge of nerve cells projecting from either side into the fourth ventricle and part of the alar plate of the afterbrain. Because of its constant relation throughout the entire vertebrate phylum, and also because of its position in the bulb, it is here called the cerebellum bulbare. Through the eighth cranial nerve it receives a large collection of fibers coming directly from the semi-

Fig. 2.—Ammocoetes, larval form of petromyzon as it is during the period when it remains embedded in the mud.

circular canals. Some fibers also enter it from the lateral line organs, and probably some—although this point has not been determined with full satisfaction—from the muscle plates. At this early stage, therefore, the ammocoete maintains its optimum physiologic position in response to afferent impulses which come from the extremely sensitive semicircular canals, from the lateral line organs and probably from the muscles. These impulses enter the bulbar cerebellum and are then transmitted to the effectors for maintenance of equilibrium. The entire cerebellum at this period is represented by two long ridges of nerve cells, one in either wall of the fourth ventricle.

At the end of three or four years, the ammocoete emerges from its mud encasement and becomes a free-swimming fish. It changes at this period from its larval to its adult form, and during this metamorphosis acquires its buccal funnel and its curious rows of stonelike teeth in its circular mouth. The eyes, which hitherto have been atrophic, now approach the surface of the head and become functionally active. The endostylar groove is converted into the thyroid gland, and almost

immediately thereafter the animal manifests the tendencies of its parasitic life, attaching itself by means of its suctorial mouth to some other fish which it proceeds to eviscerate. At this period, when the animal requires more control of its motor activities, the two long, lateral ridges in the side walls of the fourth ventricle increase rapidly in size. At their cephalic extremities a well defined bar of cells with nerve fibers connects these two ridges across the median line above the floor of the fourth ventricle (Fig. 3).

This connecting piece which joins the two lateral ridges is called the cerebellum jugale. It makes provision for the correlation of movements on the two sides of the body. Its important physiologic significance lies in the fact that it produces the synthesis of afferent nerve impulses necessary to bilateral synergized action of the body. This structure, the cerebellum jugale, I believe, foreshadows the development of the inferior vermis in the ultimate cerebellum.

Fig. 3.—Adult *Petromyzon* showing development of circular mouth and dorsal fins. There are no lateral appendages.

From the evolutional standpoint, therefore, the organ representing the most primitive cerebellum, so far as we know, may be called the archeparencephalon (original or primitive cerebellum). It occurs in cyclostomes, including the lampreys (*Petromyzonidae*), hagfish (*Myxinidae*) and bdellostoma. It comprises two morphologic structures, the cerebellum bulbare, represented by a ridge in either lateral wall of the fourth ventricle, and a connecting or yoke piece projecting into the ventricle, the cerebellum jugale.

These facts are illustrated in Figure 4, a reconstruction of the adult petromyzon brain shown in midsagittal view. In its genesis, the cerebellum at first shows no evidence of coalescence across the midline by means of a supraventricular connecting piece. During the entire period of larval life, while the animal is lying quiescent in the mud, the cerebellar structure consists of the two unconnected lateral ridges, constituting the cerebellum bulbare. As it approaches metamorphosis,

however, a strand of nerve cells begins to migrate across the roof plate. By the time the animal becomes a free-swimming organism, it has acquired a cerebellum jugale. From its inception, the cerebellum bulbare is connected by a massive group of fibers with the semicircular canals. Some fibers also enter it from the lateral line organs. These facts, taken in conjunction with the mode of life during the larval period, would seem to indicate that the cerebellum bulbare acts as a correlating organ for impulses coming in from the right and left sides of the body, through the receptors of the semicircular canals and lateral line organs, in order to maintain the animal in a posture best suited to its progressive development.

If left to the influence of physical forces alone, the ammocoete would undoubtedly be turned over on its side, or might be forced into a position resting on its back. Either of these postures would be disadvantageous

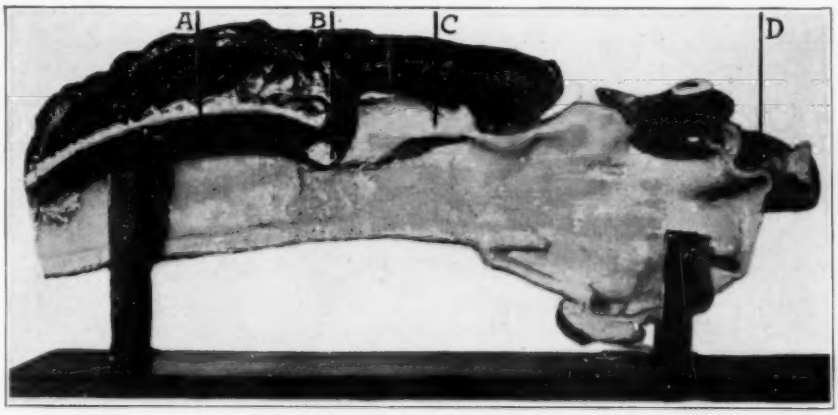

Fig. 4.—Reconstruction showing in midsagittal view the brain of *Petromyzon*. This specimen illustrates the position of the archeparencephalon (cerebellum bulbare and cerebellum jugale). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, mesencephalon; *D*, telencephalon.

in the obtaining of a food supply, or might even lead to the development of asymmetries and irregularities in the body formation. Chiefly in the interest of alimentation, then, the belly-down posture during the larval period must be accounted the best position for the animal, and this position is maintained by the mechanism provided in the presence of the lateral line organs and semicircular canals, mediating their motor effects through the cerebellum bulbare. This position of the animal which best meets its needs of life may properly be designated the optimum physiologic posture.

Immediately, however, on leaving its larval period and becoming a free-swimming fish, the animal is confronted with new problems. It

now has the more plastic environment of the water in which it must maintain its optimum physiologic posture. This posture is still that of the belly-down position, both in rest and in motion. It would now require the closer cooperation of the two sides of the body, and at this period, therefore, in response to this need, the cerebellum jugale probably made its appearance, connecting the two lateral ridges of the cerebellum bulbare across the midline.

The cerebellum, thus constituted, appears to be related to the most primitive portion of the motion-producing apparatus; namely, the archekinetic system, a mechanism which depends on relatively simple associated reflex arcs, and which is sufficient for the production of the animal's extremely limited range of movements.

In its histogenesis, the archeparencephalon, as studied in the ammocoete and petromyzon, shows a tendency of the cells aggregated in the dorsal portion of the alar plate to become more and more definitely isolated as a special localized area in the medulla into which enter the fibers from the semicircular canals and other proprioceptive organs of the body. From the definite connection of this part of the neuraxis with these proprioceptors, more particularly the semicircular canals, the inference seems justified that this region of the brain is primarily involved in some function pertaining to the proper maintenance of posture. This inference requires much further substantiation by means of experiment and other studies, but it is offered as the best hypothesis to be advanced on the evidence now in hand.

Histologically, the differentiation which takes place in these localized areas of the bulb forming the cerebellum bulbare and the cerebellum jugale, shows a tendency of a certain number of cells to assume large proportions and to be scattered diffusely in a dense matrix of smaller cells. No definite Purkinje cells or layer of cells may be discerned in *Petromyzon*. In fact, the differentiation into layers which is the characteristic feature of the cerebellum in all orders of vertebrates, does not appear distinctly in any of the cyclostomes. The tendency toward this stratification, however, is far enough advanced to foreshadow the ultimate arrangement of cellular elements in the vertebrate cerebellum (Fig. 5).

In concluding the consideration of the probable morphologic and physiologic conditions which established a demand for the cerebellum, the facts clearly point to the new mechanical condition impressed on the vertebrate by the alteration in the relation of the nervous system and the digestive tube as compared with the invertebrate. This new relation required a definite motor mechanism to maintain the optimum physiologic posture. The mechanism had its peripheral representation in the development of the lateral line organs and the semicircular canals, and its central representation in the archeparencephalon.

THE CEREBELLUM AS A POSTURE-MAINTAINING ORGAN

Accepting this conception of the cerebellum as an organ which differentiated for the primary purpose of maintaining posture, it is not difficult to perceive how, by expansion and accession, it took on itself the posture-maintaining function in all activities to which posture is essential.

Our idea of what posture is has been materially extended by the thought and researches of the last few years. Posture is not merely a requisite in the maintenance of the optimum position. Posture must be maintained through all motor action, for motion is actually a steady stream of fluid postures, just as moving pictures are produced by a steady stream of serial poses. In some previous work of the writer,

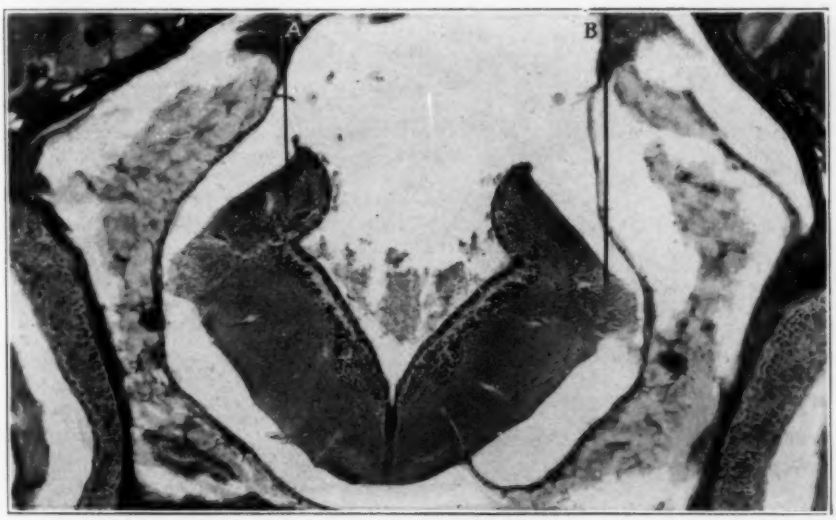

Fig. 5.—Cross section through the bulb of *Petromyzon* showing cerebellum bulbare (archeparencephalon). *A* indicates the cerebellum bulbare; *B*, the eighth nerve.

in collaboration with Dr. S. Philip Goodhart,[5] it was clearly shown by means of the ultrarapid moving picture camera that every motion is composed of a consecutive series of postures which flow one into another as the movement is produced. The analysis of each movement demonstrates that the action begins from an initial posture and ends in a terminal posture. Between these two limits is a complete series of intermediate postures. A movement may be arrested at any point in

5. Goodhart, S. P., and Tilney, F.: Analysis of Somatic Motor Disturbances. Analysis of Motor Disorders by the Aid of Ultra-rapid Moving Pictures, Neurol. Bull., **3**, Sept.-Oct., 1921.

its production between its inception and its termination, with the result that the part involved in the performance will manifest one of the component postures whose summation constitutes the act in question. Thus, raising the arm to point with the finger at some distant object, is an act comprising a complete series of postures, beginning with an initial stage, running one into another until the motion is produced and the terminal stage reached. All of the postures between the initial and terminal phases constitute a steady succession, flowing in the most direct manner to the accomplishment of the purpose. If the muscles producing this movement did not cooperate properly one with another, if the flexor groups were not properly adjusted to the extensor groups, distortions would occur in the motion and its production would not be the most direct or perfect execution from start to finish.

The matter of posture, therefore, is equally as important to action as it is to rest. Ramsay Hunt [6] has made the statement that posture follows motion like its shadow; and as a pioneer conception this figure is convenient and welcome. But posture is much more intimately concerned with motion than even this would imply. It is incorporated within the very substance of motor activity and is not in any strict sense its mere shadow.

The motion program which embodies the purposes and designs the execution of motor performance, it is generally accepted, has its inception in the forebrain; while the posture pattern necessary to such a movement arises in the cerebellum. These two streams of motor innervation must run concurrently if motor performances are to be most direct and effective in their execution.

When motor activity became more and more complex, as for example, in passing to the next higher order of vertebrates—the selachians—there appeared in such forms as the sharks and rays a much greater capability of motor performance than in the simpler cyclostomes. The motor capacity of the sharks gave rise to much more extended and much more complex movements. Their great speed in swimming, their capabilities in pursuit and attack of prey, their ability to cruise about in the water for great distances, as well as the remarkable speed with which they may rise to the surface or propel themselves to the depths, all stand out in marked contrast to the limited and relatively simple motor performances of the lampreys and hagfish. This greatly increased motor capacity is clearly indicated in the expansion of the entire central nervous system, but nowhere is it more clearly emphasized than in the development of the cerebellum. That organ has obviously kept pace with all of the new accessions of motor activity

6. Hunt, J. R.: The Statesthetic and Kinesthetic Components of the Afferent System, Arch. Neurol. & Psychiat. **8**:311 (Sept.) 1922.

seen in the sharks and rays. Complex automatic associated movements of paired lateral fins, of dorsal fins and body segments with the tail, make possible a wide increase of motor performances, not only in the selachians, but in all true fish.

The posture-maintaining organ, in adjusting itself to this increased complexity of motion, has developed a new part of the cerebellum which does not appear in the lower vertebrates. Figure 6 illustrates the

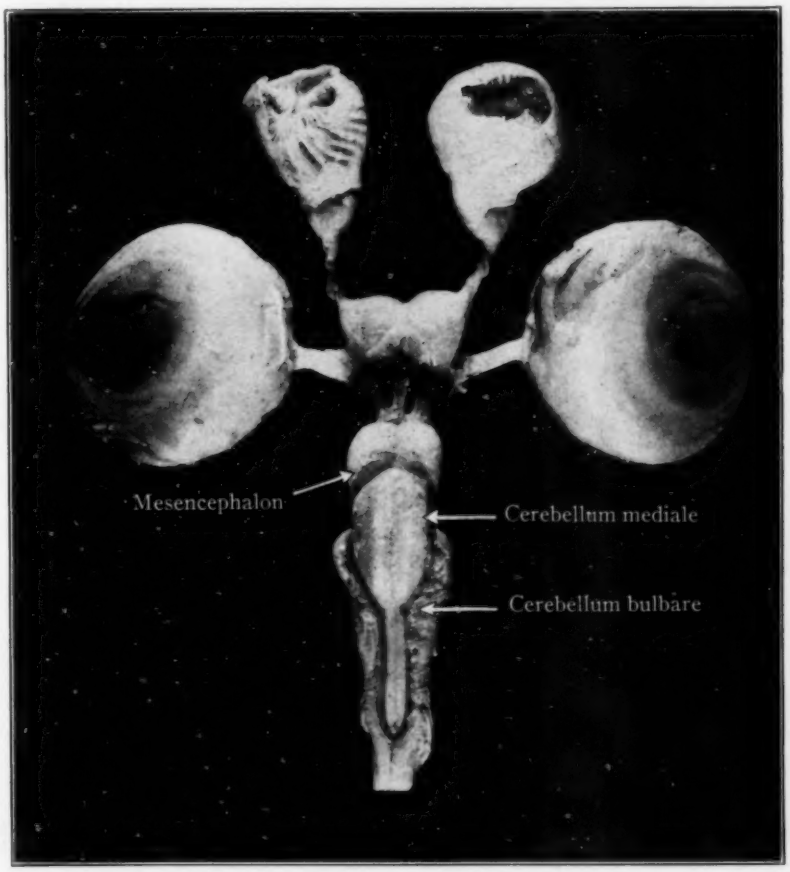

Fig. 6.—Dissection of an adult dog fish brain with eyes and olfactory organs attached, showing cerebellum bulbare and cerebellum mediale.

appearance of this new cerebellar element in one of the dog-fish (*Squalus acanthis*). It is a prominent accession to the cerebellar structures. The more primitive cerebellum bulbare may be observed much expanded in all its dimensions on either side of the medulla and extending forward toward the mesencephalon. It still occupies a prominent position in the dorsolateral wall of the medulla, and is even more

conspicuous than in *Petromyzon*. The cerebellum jugale connects the cephalic extremities of the two lateral ridges forming the cerebellum bulbare; it is now concealed by the superposition of the new cerebellar element which is here called the cerebellum mediale.

A cross-section of the dog-fish brain is shown in Figure 7. In it will be seen the new accession to cerebellar organization, the cerebellum mediale, lying ventral to which is the cerebellum jugale and flanking it on either side the two lateral ridges constituting the cerebellum bulbare.

Nothing resembling the cerebellum mediale is to be found in the lowest of the vertebrates, namely, the cyclostomes.

It is worthy of note that the cerebellum bulbare, as well as the cerebellum jugale, in all of the dog-fish has undergone a considerable expansion. Both of these parts of the primitive cerebellum have increased in size and complexity. Both now contain a granular and molecular layer. Large Purkinje-like cells are already present, arranged

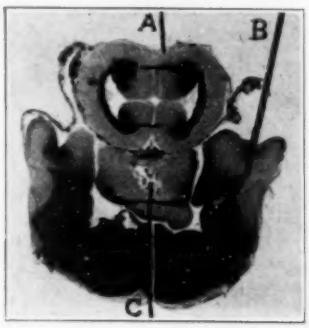

Fig. 7.—Cross section of the cerebellum in the dog fish. *A* indicates cerebellum mediale; *B*, cerebellum bulbare; *C*, cerebellum jugale.

in irregular rows on the boundary line between the granular and molecular strata. The reason for this marked increase and extensive differentiation in the archeparencephalon is undoubtedly to be found in the fact that the semicircular canal apparatus has greatly increased in size and complexity. Not only has a third semicircular canal made its appearance, but a complex appendage to the vestibular mechanism—the ductus endolymphaticus—has also developed. This addition to the labyrinth consists of a long tubular structure connected centrally with the atrium of the otocyst and extending to the surface of the head, where it becomes much convoluted and separated from the exterior by a relatively thin membrane. The ductus endolymphaticus appears embryologically in all vertebrates above and including the selachians, but in none does it reach this marked degree of complexity in its development. In the sharks it appears to be an organ closely related with the equilibrating apparatus, and from the position of its distal

extremity would seem easily accessible to the influences of pressure, depending on the depth of the water in which the animal swims. Its appearance in this complex form in the sharks undoubtedly explains in very large measure the pronounced differentiation occurring in the cerebellum bulbare.

There seems to be no reason to believe, on the grounds of anatomic evidence, that this increased vestibular complexity is in any way responsible for the appearance of the new element in selachians, namely, the cerebellum mediale. This structure is quite independent of any connection with the semicircular canals or lateral line organs. The fifth, eighth and tenth nerves, which bring in most of the fibers from the proprioceptive organs of the lateral line and semicircular canals, enter directly into the cerebellum bulbare, and many of them are seen streaming across the midline to terminate in the cerebellum jugale. None of these fibers has communication with the cerebellum mediale. From this fact, it is concluded that some reason other than the amplification in the equilibratory mechanism must explain the appearance of the new cerebellar structure. Its histology is similar to that of the cerebellum bulbare and the cerebellum jugale. It consists of a molecular and granular layer and contains cellular elements which in their position and character may be regarded as representatives of Purkinje cells. The cerebellum mediale encloses a large dorsal extension of the fourth ventricle, having a cavity of its own beneath whose ependymal lining run the heavy masses of fibers entering and leaving the organ. The connections established by these fibers, however, so far as they may be traced, indicate that this cerebellum mediale receives afferent impulses from the spinal cord, and in addition has an extensive communication with the midbrain and basal portion of the forebrain.

First inspection of a shark's brain might give the impression that the cerebellum was entirely represented by this medial structure. That such is not the case may be experimentally determined. The writer, in conjunction with Professor Elsberg, operated on a number of dogfish, detaching and completely removing the cerebellum mediale. The experiment animal was then replaced in the aquarium tank and its actions observed. The results were impressive, particularly because the animal did not manifest the amount of motor disturbance which had been anticipated from an operation causing the removal of the cerebellum. As a matter of fact, this experiment, repeated a number of times, showed clearly that the animal was well able to maintain its optimum physiologic position. It swam and rested belly down. Many of these animals lived for several days, and at no time did they manifest any tendency to lose the ability for maintaining this optimum posture. In every case, however, there was a peculiar change in the animal's

power of propulsion in swimming. It seemed to have lost the ability to coordinate the intersegmental body movements and progressed through the water wholly by means of its tail, very much as a boat is sculled by an oar from the stern. It differed entirely in its action from the swimming movements of the normal animal. The paired fins were used little, if any, and the rhythmical series of intersegmental body movements seemed to be entirely absent. If the animal was turned over on its back in the water, it immediately righted itself. If touched with a stick while resting, it would immediately swim forward, usually in a straight line, but in a more sluggish and uncertain manner than the normal animal when similarly treated. It showed no tendency to tip or turn while swimming, but proceeded with a manifest certainty maintaining its optimum physiologic position.

These experiments demonstrated that the cerebellum mediale was not connected with the motor apparatus necessary to maintain the posture best suited to the animal's purposes. They contrasted strikingly with the results obtained by Dr. Frederick Lee,[7] who experimentally removed the semicircular canals and other portions of the vestibular apparatus in sharks. As a result the animals at once showed marked distortion in the function of maintaining the optimum physiologic posture. In swimming, depending on which canal was removed, they either turned around and around on their long axes, or tail over head, rotating about their transverse axes. This would seem to indicate that the semicircular canal apparatus is definitely related to the maintenance of the optimum physiologic posture. All of its connections are with the archeparencephalon (cerebellum bulbare and cerebellum jugale).

Our own experiments, revealing as they do a lack of all such disturbance, indicate that the new part of the cerebellum, cerebellum mediale, is active in some other function, which we have interpreted as the synergizing of intersegmental body movement with the lateral paired fins and the movements of the tail.

These complex activities essential to the extensive locomotor acts of the sharks and all true fish, represent a series of automatic associated movements which bring into action the metameric series of body segments, with the paired lateral fins and the tail. In these movements, as in all other purposive acts, the two essential streams of innervation may be detected: first, that which represents the purpose and design of the movement, and second, that which controls the posture pattern necessary to its execution.

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7. Lee, F. S.: A Study of the Sense of Equilibrium in Fishes. Part 1. J. Physiol. **15**: No. 4, 1893; Part 2, ibid. **17**: Nos. 3 and 4, 1894. The Functions of the Ear and the Lateral Line in Fishes, Am. J. Physiol. **1**: No. 1, 1898. Ueber den gleichgesichtsinn, Zentralbl. f. Physiol., Nov. 19, 1892.

In selachians, therefore (sharks and rays), a new part of the cerebellum over and above the archeparencephalon already described in cyclostomes, makes its appearance. This structure is intimately connected with some function other than that of maintaining the optimum physiologic posture. It is my present belief that the cerebellum mediale from the evolutional standpoint represents what may be called the paleoparencephalon. It appears as the most conspicuous cerebellar element in all of the true fish, reptiles and birds. From the physiologic point of view, it is representative of the paleostatic element in the cerebellum, just as the cerebellum bulbare and cerebellum jugale represent the archestatic element in the most primitive vertebrate brains. This paleostatic function is concerned with the synergy of segmental (metameric) and segment (limbs, including fins, paddles and wings) musculature in the maintenance of concurrent postures necessary to inherent automatic associated movements. This paleostatic function is

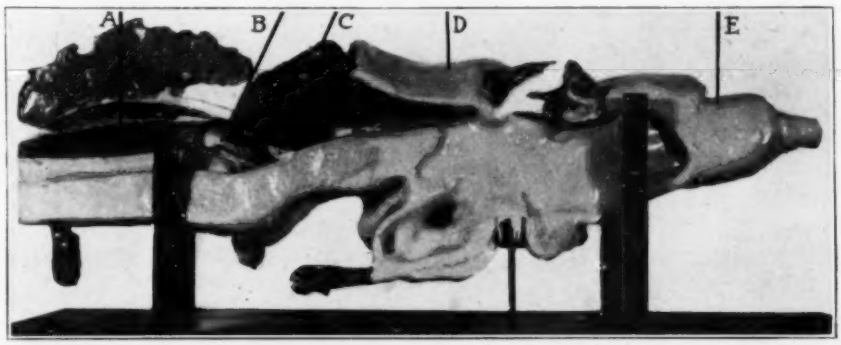

Fig. 8.—Reconstruction showing in midsagittal view the brain of a ganoid fish (*Amia calva*). This specimen illustrates the position of the archeparencephalon (cerebellum bulbare and cerebellum jugale) and the paleoparencephalon (cerebellum mediale). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, mesencephalon; *E*, telencephalon.

related to the paleokinetic system which, according to our present interpretation, has to do with the regulation of inherent automatic associated movements.

In Figure 8 is seen a reconstruction in midsagittal view of a brain in a ganoid fish (*Amia Calva*) in which the archeparencephalon and paleoparencephalon are shown. Figure 9 shows a dissection of the brain of a codfish, while Figure 10 represents a cross section of the brain of a codfish, showing the cerebellum bulbare, cerebellum jugale and cerebellum mediale.

An interesting sidelight on the progressive evolution of the cerebellum is afforded by the amphibia. The assumption of an amphibious

character of life with the partial establishment of a terrestrial habitat, appears in its inceptions to have concerned itself mostly with the adaptations to living on land. These adaptations were simple in the extreme and the motor capacities of the animal were limited, perhaps for lack of experience and opportunity, in their new environment. That the

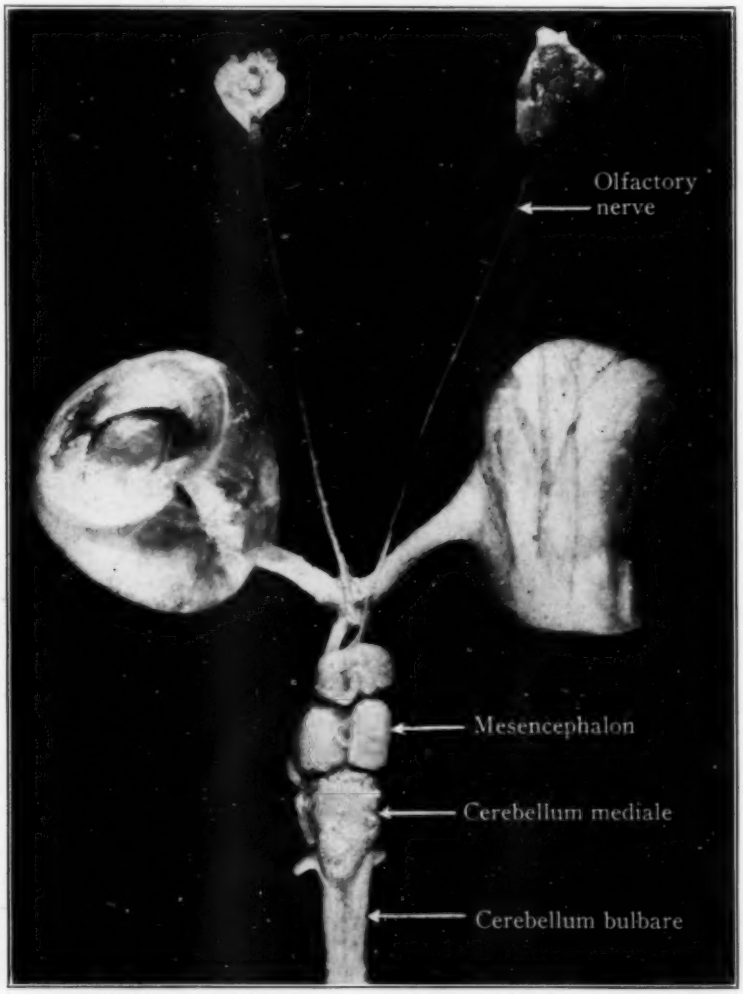

Fig. 9.—Dissection of cod fish brain with eyes and olfactory organs attached, showing cerebellum bulbare and cerebellum mediale.

transition from water-living to land-living was at once a decisive one probably does not express the fact. During the assumption of terrestrial life the process of transition was probably a slow one, taking the animal gradually out of the water to the soft marsh lands, until finally it was

able to maintain itself definitely on dry land. This intermediate period carried out in the marsh lands, provided an environment in which it was difficult to develop complex motor activities. It is for this reason that the amphibia in general, and particularly the anura (frogs and toads) have differentiated to such a slight degree in their cerebellar organization. They laid the foundations for life on dry land and were an extremely generalized type out of which terrestrial vertebrates took origin.

Fig. 10.—Cross section of the cerebellum in the cod fish. *A* indicates the cerebellum mediale; *B*, cerebellum bulbare; *C*, cerebellum jugale.

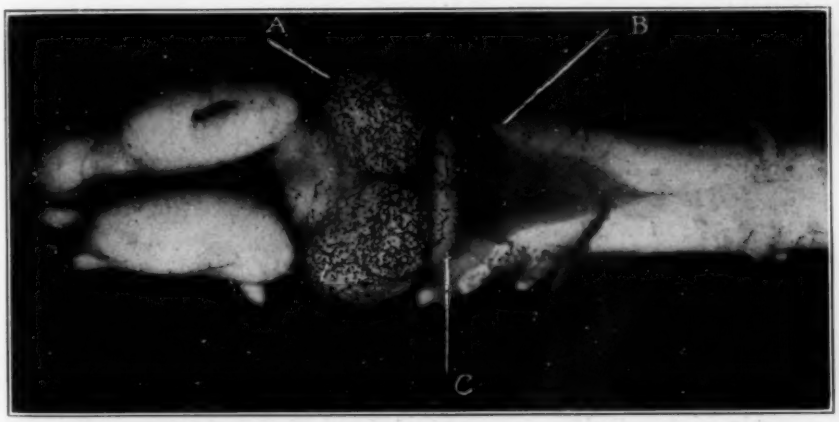

Fig. 11.—Dissection of frog's brain showing cerebellum bulbare and cerebellum mediale. *A* indicates mesencephalon; *B*, cerebellum bulbare; C, cerebellum mediale.

Figure 11 shows the cerebellum of the frog (*Rana sylvatica*). In cross section this cerebellum reveals the archeparencephalon (cerebellum bulbare and cerebellum jugale) with a small dorsal expansion representing the paleoparencephalon (cerebellum mediale). The simple

development of the cerebellum in amphibians has been a stumbling block in the explanation of the progressive evolution of the brain. When, however, the limited motor activities of amphibia in general are considered, it becomes clearer how their generalized cerebellar pattern was necessary as a stepping-stone for further advances in evolution.

In the advancement to the age of reptiles, little or no positive gain was made in increasing the capacity of locomotion. Whatever may

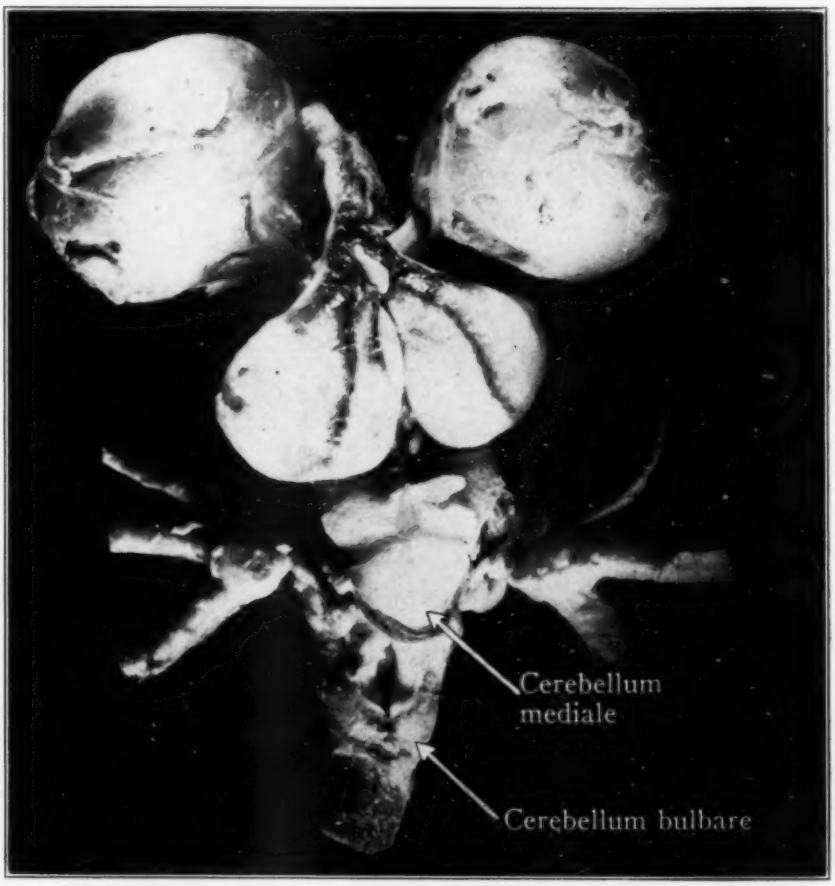

Fig. 12.—Dissection of alligator brain showing cerebellum bulbare and cerebellum mediale.

have been the character of the cerebellar organization in the gigantic tetrapod reptiles that inhabited the earth during the mesozoic age, it is certain that their modern congeners show little of progress in cerebellar evolution over the fish. The reptilian cerebellum, although it presents the three parts already observed in vertebrates lower in the scale, has expanded little as compared with the sharks.

Figure 12 shows a dissection of an alligator brain, and gives a good idea of the degree of development attained by reptiles generally speaking.

The next great step forward occurred in the birds, when a terrestrial habitat had become fully established. The development of the wings for flying and the necessary biped locomotion in consequence of this high specialization of the fore limbs introduced new problems in motion. In the main, the use of wings differs only in degree of complexity from the use of fins or paddles. The alar oscillations in flying are most delicately adjusted. The posture patterns of such complex automatic associated movements as those of flight require a corresponding complexity in the cerebellum mediale. This complexity is witnessed in the bird by the elaborate foliation of the cerebellum. In alighting, as well as in starting to fly, the bird has new problems of equilibrium. Its motor adjustments are even greater during locomotion on the ground. In the main, the actions of the hind limbs represent automatic associated movements of biped locomotion. On the other hand, most birds, and especially the ratite (running) birds, such as the ostrich and cassowary, have developed a high degree of independent action in each hind leg. They are able to use the claws in scratching for food and even to strike heavy blows with one leg while standing on the other. In certain birds, this same ability is witnessed in the wings, which are used in defense and attack. For the most part, however, the independent striking power of each wing is limited and probably belongs much more definitely to the type of automatic associated movement than to unilateral independent action.

It would seem, therefore, that in the birds the highest degree of unilateral independent action has been acquired in the legs, and this is probably reflected in a new element appearing for the first time in the avian cerebellum. A small lateral evagination from the cerebellum mediale, extending over the lateral aspect of the bulb on either side, marks the beginning of the cerebellum laterale. Many authorities have believed that this lateral extension of the cerebellum is the forerunner of the flocculus and is related to the movements of the tail. If such were the case, it might reasonably be expected that the cerebellum in all fish whose tail movements are complex should also have this lateral extension. The same objection would apply in forms like alligators and crocodiles; in fact, in all reptiles which use the tail extensively in locomotion. Comparative morphology does not lend support to the conception that this lateral extension of the avian cerebellum is concerned with the coordinative control of the tail. It is my opinion that these lateral expansions which appear in the later stages of embryonic development in birds as two lateral evaginations, represent entirely new elements in the cerebellar organization. They indicate the beginning of cerebellar control of unilateral independent movements in the legs.

The cerebellum of one of the running birds—the Australian ostrich —is shown in Figure 13. A cross-section of this brain in Figure 14 represents the compound nature of the organ, according to the lines already laid down. It illustrates the presence of the cerebellum bulbare, cerebellum jugale, cerebellum mediale, with the addition of this new part, the cerebellum laterale. The latter structure reaches

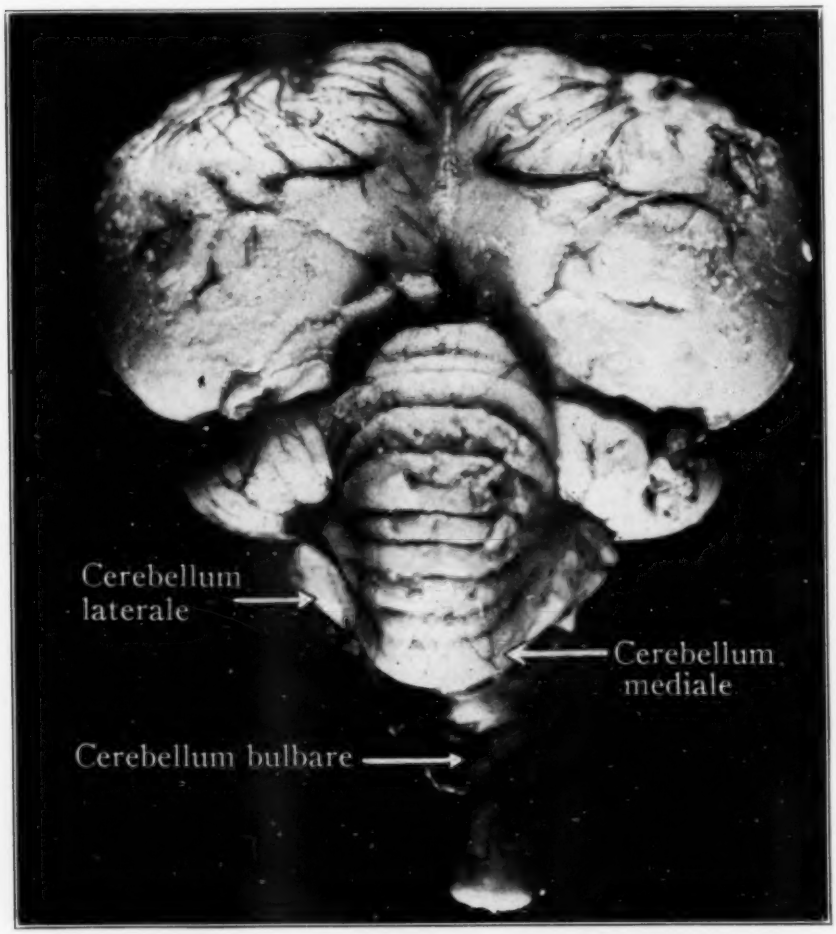

Fig. 13.—Dissection of emu brain (Australian ostrich) showing cerebellum bulbare, cerebellum mediale and cerebellum laterale.

its greatest development in the lateral lobes of the cerebellum in animals which have acquired the most highly differentiated fore limbs, whose distal extremities have developed as hands, and whose hind limbs serve the purposes of the most complete bipedal locomotion.

From the evolutional standpoint, the cerebellum laterale represents the neoparencephalon. It rapidly expands in the different orders of mammals and reaches its highest development in the anthropoids and man. That the influences which underly its existence are directly connected with the differentiation of the fore and hind limbs, is shown by the progressive increase of nerve fibers entering the inferior cerebellar peduncle. Still further evidence is furnished by the development in the mammal of the middle cerebellar peduncle, which contributes its entire collection of fibers to the lateral cerebellar lobes. This peduncle is an index of the degree of connection between the cerebral cortex and the cortex of the cerebellum. It increases in volume in direct ratio to the

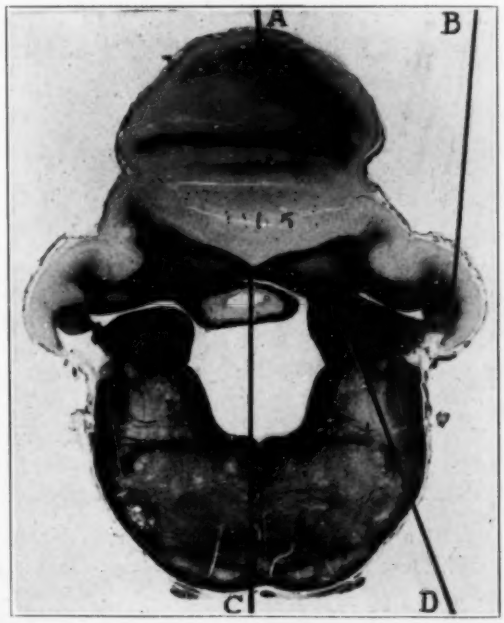

Fig. 14.—Cross section of the cerebellum in the emu (Australian ostrich). *A* indicates cerebellum mediale; *B*, cerebellum laterale; *C*, cerebellum jugale; *D*, cerebellum bulbare.

increase of the cerebral cortex, and attains its greatest dimensions in animals who have the most perfectly developed hands.

From the physiologic point of view, the neoparencephalon represents the neostatic cerebellar element. It is concerned with the synergy of the limb musculature necessary to the maintenance of the concurrent postures in acquired independent movements. The arms and legs in man and the anthropoids still retain many of the ancient automatic associated movements which are inherent. Their outstanding characteristic, however, is the fact that they are capable of developing many

complex movements which are acquired through constant repetition and concentration of attention. These skilled acts depend on the teachability of the cerebral cortex. They are highly individualistic rather than generic, and represent the ultimate independence of action in the limbs on the two sides of the body.

THE MAMMALIAN CEREBELLUM

A comparative study of the mammalian brain most convincingly reveals the relation of this capacity in skilled movement to the development of the cerebellum laterale. In the rabbit (Fig. 15) the lateral lobes are present but show a slight expansion only, in direct proportion to the degree of independent movement which this animal has acquired in the fore and hind limbs. The extremities are still used largely in the automatic associations of locomotion. This same observation applies

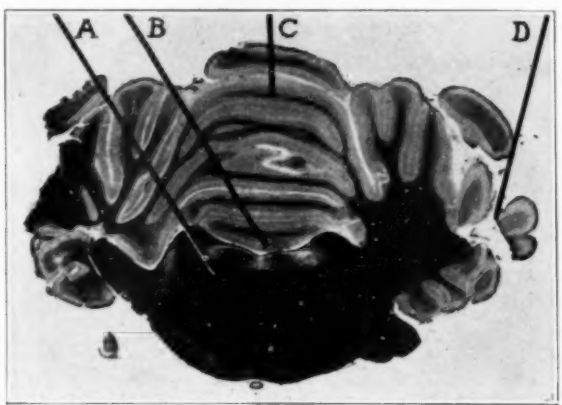

Fig. 15.—Cross section of the cerebellum in the rabbit. *A* indicates the cerebellum bulbare; *B*, the cerebellum jugale; *C*, the cerebellum mediale; *D*, the cerebellum laterale.

to all rodents, to ungulates and even to carnivores, although in the cat family there is a decided increase in the lateral cerebellar lobes in direct proportion as these animals have acquired more independent use of the fore limbs. In the transition from the carnivores to the primates, the lemur probably represents an intermediate stage. This animal shows an even more marked increase in its lateral cerebellar lobes than does the cat. It is still, however, a distinctly quadruped animal, and has freed its fore limbs but little for purposes other than locomotion. The cerebellum of the lemur is seen in Figure 16.

Very decisive is the advance observed in one of the new world monkeys (*Mycetes sineculus,* Fig. 17). This animal has acquired a considerable degree of independent skilled use of the hands.

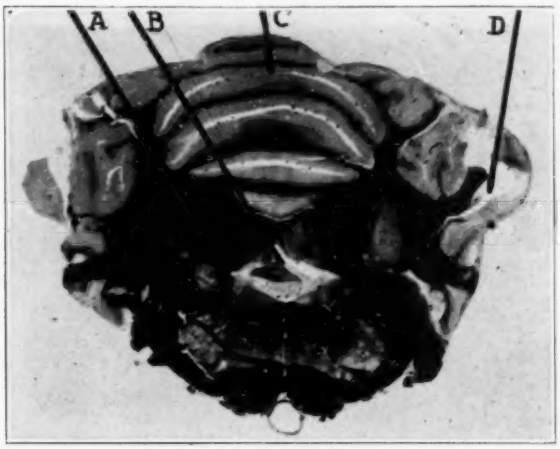

Fig. 16.—Cross section of the cerebellum in lemur. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

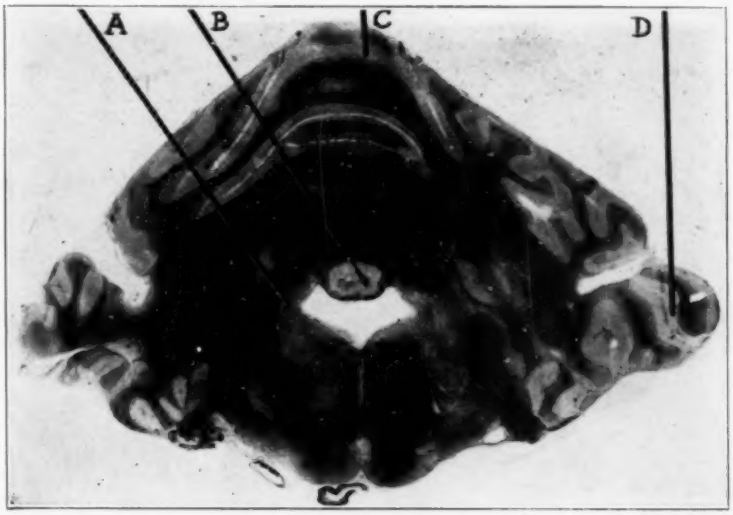

Fig. 17.—Cross section of the cerebellum in a South American monkey (*Mycetes sineculus*). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

In the gibbon (Fig. 18), an early derivative of the common primate stock, the lateral lobes of the cerebellum have expanded still more. This animal is noted for its long arms and its well differentiated hands. These it uses not only for purposes of locomotion, but in many acquired skilled acts pertaining to defense and to the efforts of obtaining food.

In the three great anthropoids, orang-utan, chimpanzee and gorilla (Figs. 19, 20 and 21), the marked expansion of the lateral lobes of the cerebellum is clearly evident. The progressive approach to bipedal locomotion is well recognized in these animals, together with the freeing of the fore limbs for other purposes. How much the chimpanzee, and even the orang-utan, may be taught in the way of skilled movements with the arms and hands, is a matter of familiar knowledge. The

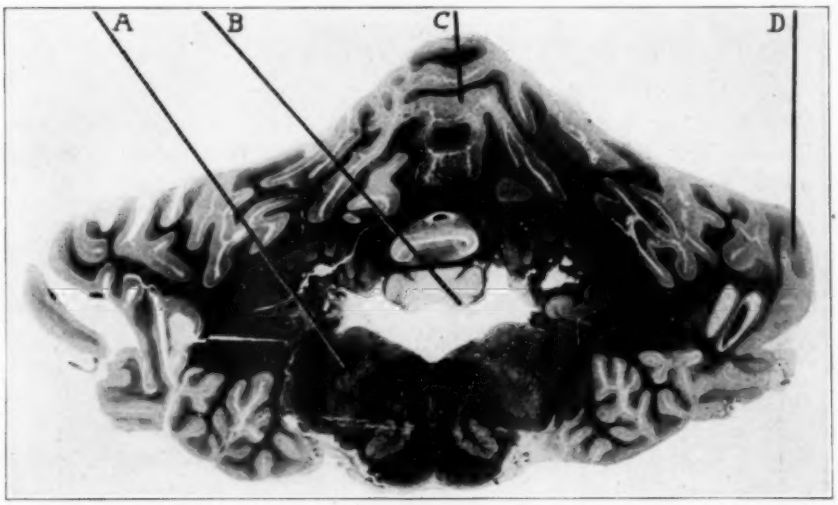

Fig. 18.—Cross section of the cerebellum in the gibbon. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

history of the gorilla, John Daniel, whose brain I am at present studying, affords an excellent example of the teachability of the anthropoid cortex. This animal was almost human in its manual achievements. The gorilla cerebellum here shown is that of Dinah, who died some time ago in the New York Zoological Gardens. This animal was only 3 years old, but had been taught to employ her hands with considerable skill, using a knife and fork, drinking from a cup and playing with a ball.

The final proof of the relation between the lateral lobes of the cerebellum and independent skilled movements, especially of the hands, is afforded by the human brain. A section of the cerebellar region in man is shown in Figure 22.

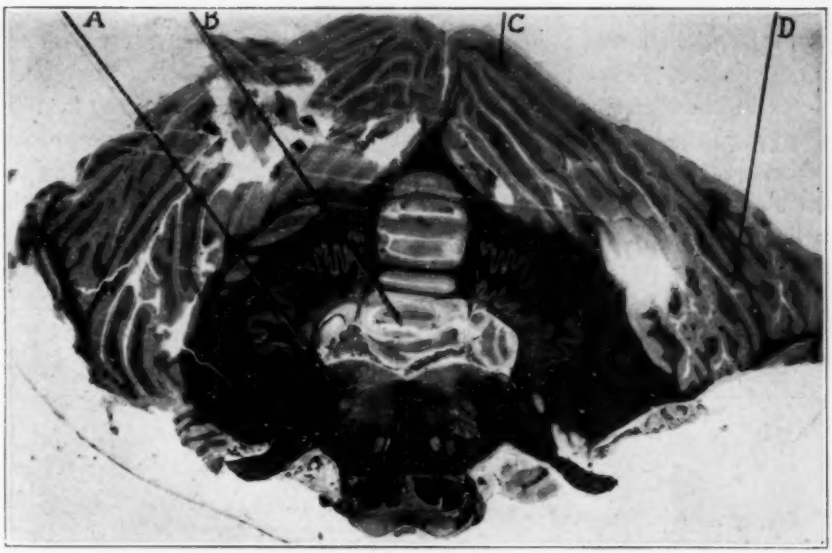

Fig. 19.—Cross section of the cerebellum in an orang-utan. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

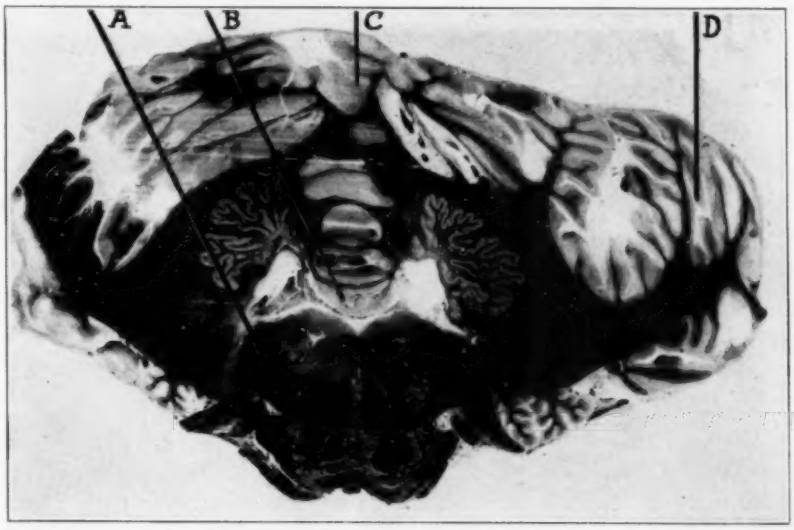

Fig. 20.—Cross section of the cerebellum in a chimpanzee. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

Such evidence as that afforded by the primates seems to substantiate the proposal that the neoparencephalon made its appearance in response to the development of the cerebral cortex. It seems to be directly related to the control necessary for skilled movements. Reviewing the conditions in the vertebrates, beginning with *Petromyzon* and continuing upward in the several orders of fish, it becomes apparent that the process of cerebellar expansion has passed through the evolutional phase of the archeparencephalon to the epoch marked by the acquisition of the paleoparencephalon in selachians and all true fish. Then, with the appearance of birds, the lateral lobes had their inception and became more conspicuous in passing to the mammals. Through the mammalian orders, including monotremes, marsupials, rodents, carnivores and pri-

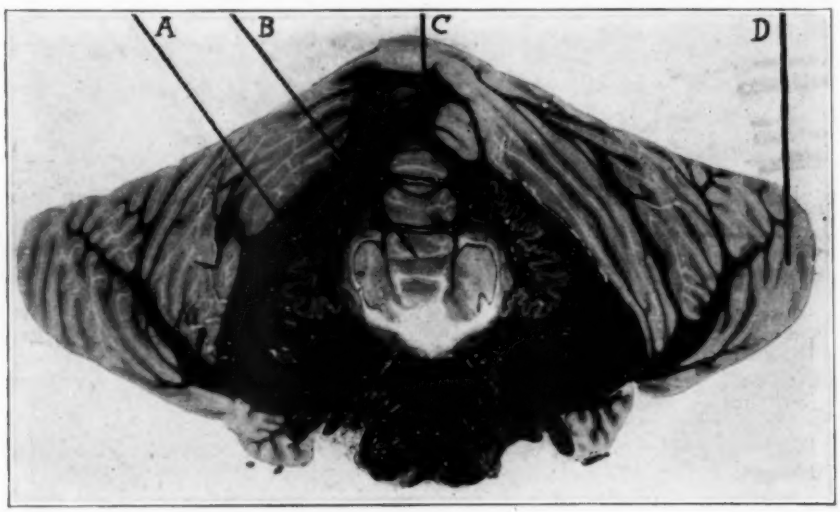

Fig. 21.—Cross section of the cerebellum in a gorilla. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

mates, the lateral lobes of the cerebellum have gradually expanded as the fore limbs have been progressively released from the functions of locomotion.

EXPERIMENTAL EVIDENCE

With the aim of controlling these tentative conclusions, several experiments were performed on animals, particularly with the idea of confirming the evolutional interpretation of the cerebellum presented here. This work was done in collaboration with Professor Pike.

The first animal operated on was a cat in which the right semicircular canals were destroyed. This operation aimed to interrupt the integrity of the apparatus connected with the archeparencephalon.

In consequence of the operation the animal was no longer able to maintain its optimum physiologic posture, although all of its movements in the effort to do so were well coordinated. It made vain attempts to get on its feet, to lie on its belly or to direct its head forward. These attempts resulted in constant but ineffective movements. This experiment illustrates what occurs when the archestatic system is disturbed.

In the second experiment, the lateral lobe of the cerebellum of a cat was destroyed on one side. Following the operation, the animal manifested an asynergy in the movements of the limbs on the same side as the operation. Although it was still able to maintain a proper physiologic posture, the movements of the hind and fore limbs on the

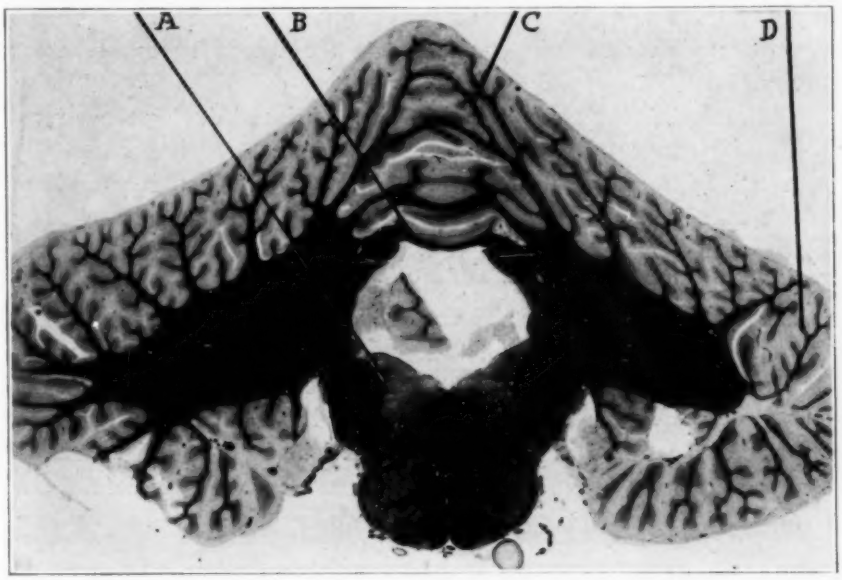

Fig. 22.—Cross section of the cerebellum in man. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

same side as the lesion showed a marked dysmetria, because the stream of postures necessary to well coordinated movement no longer ran concurrently with the motor program of the animal's locomotor acts.

These two experiments bring into contrast the effects of disturbing, first, the archeparencephalon in its function of maintaining the optimum physiologic posture, and, second, the paleoparencephalon in its capacity of maintaining the postures necessary to the automatic associated movements of locomotion.

A second set of experiments with pigeons served to emphasize these conclusions. In one pigeon the semicircular canals on the right side

were removed. This animal maintained well coordinated movements, but was unable to hold itself in proper posture. In a second pigeon, the entire cerebellum, including all of its components, was removed. In this case not only was the power of maintaining the proper physiologic posture lost, but every movement with wings, legs, neck and tail showed a complete asynergy.

SUMMARY

It is my present belief that phylogenetically the cerebellum represents an integrative process of specialization. This process, as the result of increasing motor capacity during the progress of evolution, has centralized in one organ certain specialized structures acquired from time to time as need has been. The principal purpose of this mechanism has been the maintenance of posture in all the varied types of motor activity.

The cerebellar components which have developed during the several epochs of phylogeny and which have become more or less completely integrated within the ultimate cerebellum, are: (1) the cerebellum bulbare and the cerebellum jugale, representing the archeparencephalon; (2) the cerebellum mediale, representing the paleoparencephalon; and (3) the cerebellum laterale, representing the neoparencephalon.

The cerebellum bulbare and cerebellum jugale, the most primitive components of the cerebellum, took origin in the medulla oblongata. They are the sole representatives of cerebellar organization in the cyclostomes (lampreys and hagfish). Their homologues in the human brain are to be found in the vestibular nuclei of the medulla and the inferior cerebellar vermis. In man the greater portion of the cerebellum bulbare has become incorporated in the inferior vermis. Its bulbar remnants are seen in the vestibular nuclei.

The cerebellum mediale, the next component to appear, took origin from the roof of the medulla oblongata as a single unpaired evagination. It developed as the principal addition to the cerebellum bulbare and cerebellum jugale in all true fish, amphibians, reptiles and birds. Its homologue in the human brain is to be found in the superior cerebellar vermis.

The cerebellum laterale, the last component to appear, took origin in two lateral evaginations from the cerebellum mediale, symmetrically bilateral and one on either side. It forms an expansion of the cerebellum mediale and first appears in birds, but reaches its highest development in mammals, particularly in the primates. Its homologue in the human brain is to be found in the lateral lobes of the cerebellum.

The cerebellum bulbare and cerebellum jugale developed in the vertebrate as a central controlling structure of a mechanism necessary

to maintain the optimum physiologic posture of the animal. It became essential in consequence of the altered neuro-enteric relations determined by the invertebrovertebrate transition.

Having thus established a proclivity to centralize the control of posture-maintaining function, this general region of the brain acquired by evolutional accession, the cerebellum mediale. This cerebellar component is essential to the posture-maintaining function of inherent automatic associated movements.

The cerebellum laterale developed to supply the necessary posture-maintaining function in the acquired, independent movements, especially of the arms and legs. It has reached its greatest development in man, whose capacity in acquired skilled acts with hands and feet far surpasses that of all other animals.

Each step in this evolutional process has required the passage of age-long epochs of time. The following tabulation may make more graphic the stages which have been traversed in the phylogenetic achievement of this integrative process in the cerebellum.

ANALYSIS OF CEREBELLAR COMPONENTS

EVOLUTIONAL PHASE	MORPHOLOGIC ELEMENTS	PHYSIOLOGIC PHASE
Archeparencephalon (cyclostomes, lampreys and hagfish) From Silurian to Devonian	Cerebellum bulbare Cerebellum jugale	Archestatic: Synergy of dorsal, ventral and lateral musculature in maintenance of optimum physiologic posture; related to Archekinetic mechanism
Paleoparencephalon (all true fish, amphibia, reptiles and birds) Through Devonian to Palaeocene	Cerebellum mediale	Paleostatic: Synergy of segmental and segment musculature (limbs) in maintenance of concurrent posture in inherent automatic associated movements; related to Paleokinetic mechanism
Neoparencephalon (mammals) Palaeocene to recent	Cerebellum laterale	Neostatic: Synergy of segment musculature (limbs) in maintenance of concurrent postures in acquired independent movements; related to Neokinetic mechanism

A CLINICOPATHOLOGIC STUDY OF ACUTE AND CHRONIC CHOREA*

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Our main object in presenting this paper is to report the pathologic findings in two cases of chorea, one of the acute and one of the chronic variety. Until recent years the pathology of these two conditions has been elusive, and even today we do not have a clear conception of the underlying anatomic changes of the choreas; this is especially true of Sydenham's chorea.

Until Marie and Tretiakoff, in 1920, reported their case of Sydenham's chorea there was practically no known pathology in this condition so far as the nervous system was concerned. These writers described in their case findings identical with those seen in patients who died of epidemic encephalitis. In fact, as one reads the report of Marie and Tretiakoff one wonders whether their case was not one of the choreiform type of encephalitis.

One of us saw a case in the winter of 1920-1921 which he diagnosed as a severe case of Sydenham's chorea in a boy of 14, who later in the course of his disease developed lethargy and other typical signs of encephalitis from which he made a complete recovery. Had this patient died in the first stage of the infection, he would have been considered as having had Sydenham's chorea, and yet the outcome proved beyond a doubt that it was a case of epidemic encephalitis. As further evidence that Marie and Tretiakoff's case was not one of acute chorea may be mentioned the fact that there were no signs of endocarditis.

The possible relation between chorea and encephalitis has been discussed by Harvier and Levaditi, whose conclusions are: "1. Certain acute febrile choreas are brought about by the virus of encephalitis. 2. It is not yet proved that *all* acute febrile choreas are due to this agency."

REPORT OF CASES

CASE 1.—*History.*—A patient with Sydenham's chorea was admitted to the medical service of the hospital of the University of Pennsylvania on July 4, 1920, and was assigned to the service of Dr. Alfred Stengel.

On June 19, 1920, the patient, who was a girl, 12 years of age, began to have involuntary jerkings of the upper extremities. Two or three days later

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* From the neurological service and neuropathologic laboratory of the Philadelphia General Hospital, and from the School of Medicine of the University of Pennsylvania.

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the movements involved the head and the tongue and then the lower extremities. At the time of her admission to the hospital she could not talk and was confined to bed. Physical examination revealed a well nourished child with typical choreiform movements involving all four extremities, the trunk, the head and the tongue. A systolic murmur was heard at the mitral and pulmonic areas. The blood examination showed an eosinophilia of 6 per cent.

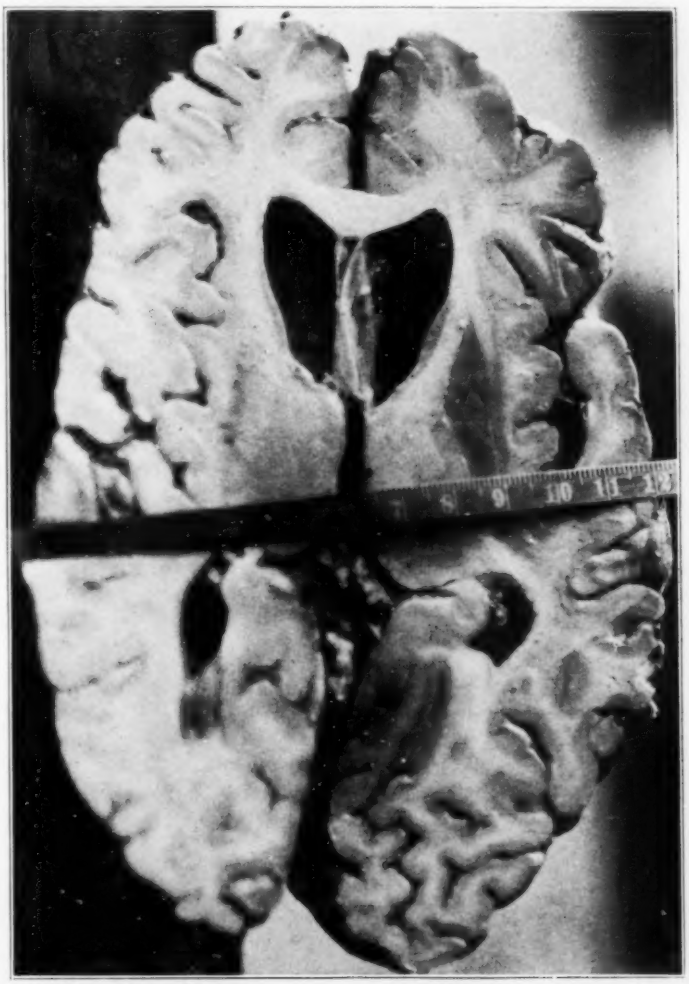

Fig. 1 (Case 2).—Horizontal section of hemispheres, showing dilatation of ventricle and atrophy of caudate and lenticular nuclei.

For six days following her admission her temperature was at or near normal, but after that it showed some variations, the temperature going as high as 101 to 102 F., and even as high as 105 degrees before death.

Necropsy Examination.—This revealed vegetations, semisoft but well attached on the auricular side of the mitral leaflets. These vegetations averaged 1 to 2 mm. in size. All the other valves were normal. A thorough and systematic

examination was made of the brain with special reference to the basal ganglions. In this study the toluidin blue, Weigert; phosphotungstic acid hematoxylin, Alzheimer-Mann, Marchi, Bielschowsky and hematoxylin and eosin stains were used. It is entirely proper to state at this point that absolutely no changes were found in the brain that could not be attributed to the acute febrile condition from which the patient suffered for some days before death. No changes were found which in any way approached the findings which Marie and Tretiakoff described in their case of Sydenham's chorea, a case which was probably one of epidemic encephalitis.

Fig. 2 (Case 2).—Basal ganglions. Note atrophic striatum, caudate nucleus and putamen and absence of internuclear fibers. *Nc.* indicates the caudate nucleus; *Put.*, the putamen; *Thal.*, the thalamus. Weigert stain.

A white man, 52 years of age, was admitted to the Philadelphia Hospital on Jan. 25, 1905, and died on March 3, 1917. His family history is one showing a marked degenerative defect as eight of his blood relations have had Huntington's chorea; other members of the family have been insane. The man's choreiform movements began in 1910, and shortly afterward he began to show mental symptoms, chiefly those of a simple dementia. The choreiform movements affected his entire body, and involved to a marked degree the muscles of his trunk and pelvis. His gait was rendered extremely bizarre by these movements, the unusual thing in his gait being a series of bowing movements. Some considered this a case of dystonia musculorum deformans.

Necropsy Examination.—The brain appeared to be what we have chosen to call "paretic looking." The cerebrum, cerebellum, brain stem and spinal cord were smaller than normal. The convolutions were atrophic and the fissures widened, this being especially evident over the frontorolandic areas. The pia-arachnoid was everywhere thickened, but to a more marked degree over the anterior half of the brain where it concealed the underlying structures by its opacity. Decortication could be performed without tearing the cortex.

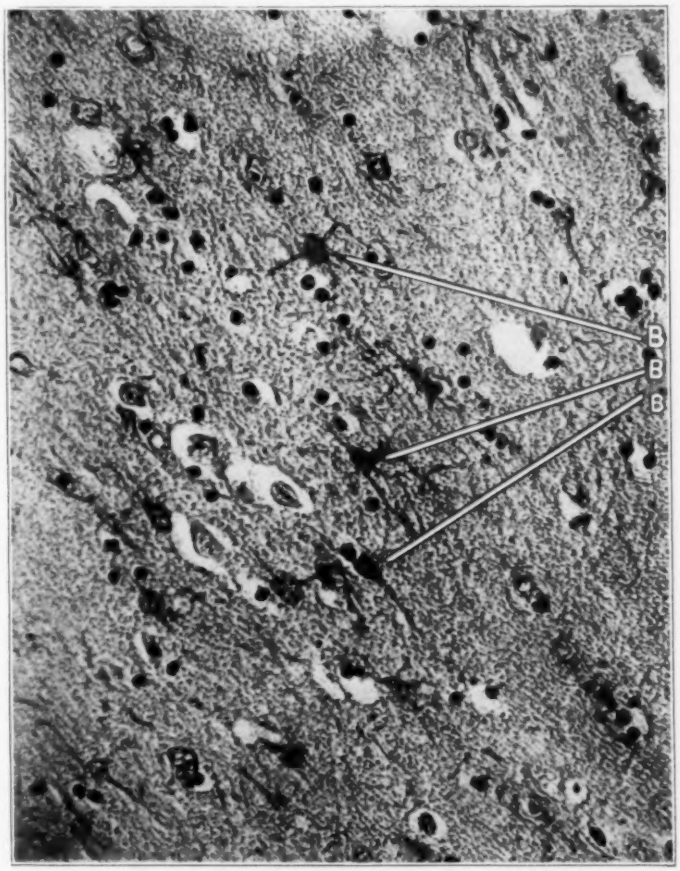

Fig. 3 (Case 2).—Putamen, showing typical astrocytes, *B*. Phosphotungstic acid hematoxylin stain; × 368.

Cross section of the brain in the usual manner showed that the ventricles were dilated (Fig. 1) and that the basal ganglions were small in proportion to the rest of the brain. The cortex was visibly narrowed, especially anteriorly.

The microscopic method of examination included large sections from different areas of the cerebrum, brain stem, cerebellum, spinal cord and serial sections through the basal ganglions and hypothalamic region. The stains used were the same as in the preceding case.

The Cortex.—Weigert stain showed atrophic convolutions separated by widened fissures and a narrow cortex with loss of many of the myelinated

fibers in all layers, most noticeable anteriorly. With toluidin blue the cyto-architecture was profoundly altered, so much so that the normal relationship could not be recognized. An occasional Betz cell remained to identify the motor area. The ganglion cells throughout, but especially in the fronto-rolandic cortex were decreased in number, and diseased. Those which remained exhibited various forms of acute and chronic cell change, such as simple chromatolysis, axonal degeneration, Nissl's severe cell disease and atrophy. The silver impregnation stains confirmed the findings obtained with toluidin blue. The vessels were not increased in number, but they showed distinct

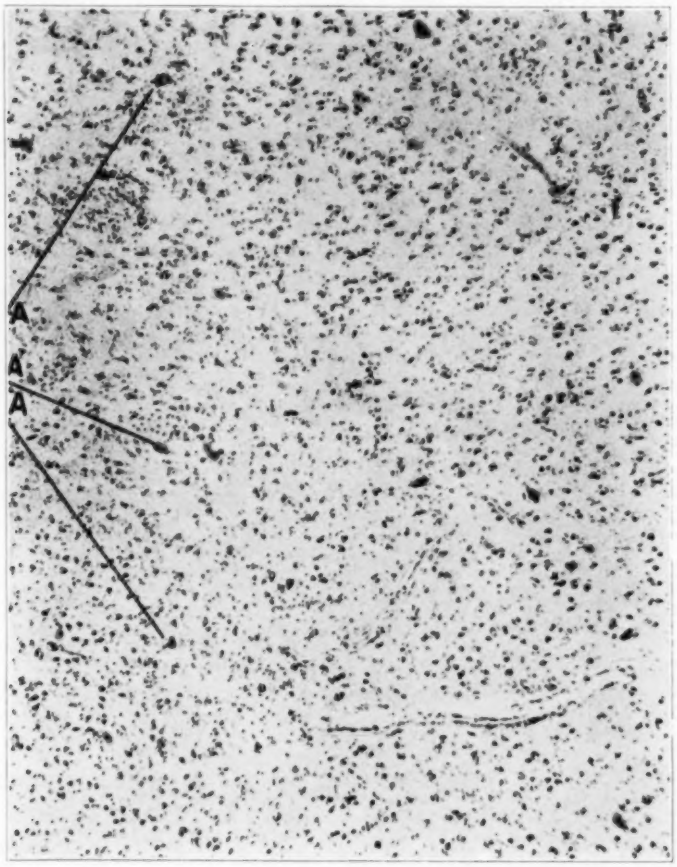

Fig. 4.—Normal putamen. Note satellitosis around large ganglion cells at *A*. Toluidin blue; × 57. (Use hand lens.)

sclerosis and hyaline degeneration; for the most part the perivascular spaces were dilated, containing only the products of brain degeneration which at first glance simulated the cuffing with lymphocytes and plasma cells found in paresis. Glia cells were increased in number.

The pia-arachnoid showed distinct thickening, due to a connective tissue proliferation and containing within its meshes cells which on differential staining proved to be gitter cells with a tendency to agglutinate in the region of

fissures and about the vessels. Amyloid bodies were present, especially in the outer layers of the cortex and in relation to the ventricles, showing that there was a slow degenerative process going on. Although sought for, no foci of degeneration were found as described by Klebs, Oppenheim and Hoppe, Marie and Lhermitte and others; nor were plaques found such as are seen in senile dementia and at times in senility. The central white substance showed atrophy of the projection system of the cortex with replacement by neuroglia fibers arranged similarly to the destroyed fibers, showing that the process had taken place slowly.

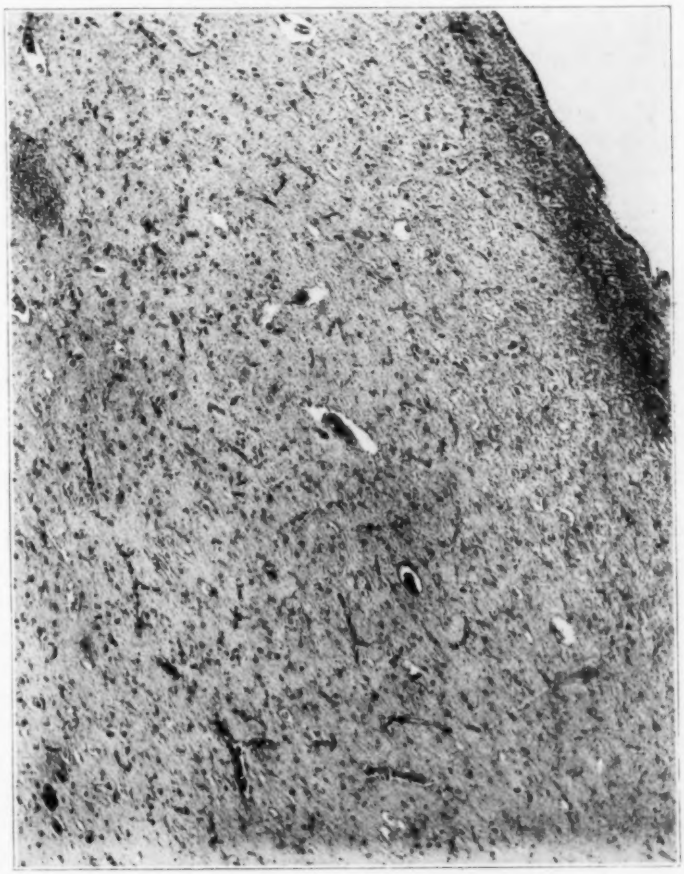

Fig. 5 (Case 2).—Caudate nucleus. Note intense glial infiltration. Phosphotungstic acid hematoxylin stain; × 57.

The Cerebellar System.—In the study of large sections of the cerebellum, including serial sections of the dentate nucleus, nothing of note was found except a smallness of all the structures. There were no areas of softening. The pontile nuclei, red nuclei and olives were intact.

The Central Ganglions.—As shown in Figure 2, with the Weigert stain there was marked atrophy of the central ganglions, particularly of the caudate and lenticular nuclei. As can be seen, the caudate was distinctly shrunken; the

putamen was apparently more involved than the globus pallidus. The thalamus was relatively intact. The internal capsule stained well, showing no areas of degeneration, and its dimensions were in proportion to the size of the brain. The internuclear fibers which take origin in the small cells of the caudate nucleus and putamen (the striatum of C. and O. Vogt) were absent. Serial sections through the hypothalamic region revealed no degeneration of the projection fibers (striothalamic, striosubthalamic and striomesencephalic radiations).

More minute histologic study showed that the nerve cells had practically all disappeared from the caudate and putamen, with only a few of the smaller and some of the larger motor type (Malone) of cells remaining. The phosphotungstic-acid hematoxylin stain showed an extreme proliferation of the glial elements, especially of the astrocytes (Fig. 3), which had a tendency to collect about the vessels. As could be seen in the gross, the globus pallidus (the pallidum of C. and O. Vogt) was much less involved. The characteristic large cells were present in the usual number. Glia cells were slightly increased, although astrocytes were rare. In the thalamus, there were atrophic lesions of the cells with a glial proliferation, but infinitely less intense than in the striatum. The vessels showed the same changes as found in the cortex—thickening of their walls, and occasional perivascular collections of degenerated material. No foci of degeneration were seen.

The spinal cord showed no pathologic condition, and the liver, except for congestion, was normal.

COMMENT

The changes found in the case of Huntington's chorea are distinct and rather widespread, involving the frontorolandic cortex, the meninges, the vessels, the caudate nucleus and putamen, with an escape of the globus pallidus and the optic thalamus. It is worth noting at this point that despite the extensive cortical and striatal changes, no secondary degeneration was found in the pyramidal tracts or in the extrapyramidal system. The fact that the caudate and putamen are selected in this disease with an escape of the globus pallidus is easily explainable when it is realized that the striatum is developed from the same part and is really one body separated in man by the passage of the anterior fibers of the internal capsule. As is well known, the globus pallidus is composed of large ganglion cells, which Malone has shown to be related to the motor system in their general structure. Some of these cells are scattered in the caudate and putamen, and they are relatively uninvolved. It is the small type of cell which bears the brunt of the pathologic process.

We were disappointed in the absence of pathologic findings in the case of acute chorea, especially so because Marie and Tretiakoff found such marked alterations in their case. Our case was undoubtedly one of Sydenham's chorea, the diagnosis being based on the type of irregular movement seen, the presence of endocarditis and eosinophilia. The case was not one of encephalitis, because the lesions of that disease were absent. We believe that in the course of Sydenham's chorea cerebral changes must occur, but modern methods have so far failed to

reveal them. Care must be taken in the interpretation of the various sections taken from the basal ganglions, because in control sections made from relatively normal persons the same cell changes were demonstrated as in the case of Sydenham's chorea. As Bielschowsky has shown, the collection of cells around the large motor type of ganglion cell in the corpus striatum will occur under normal conditions, an indication of the fragility of these elements. This was found in the case of Huntington's chorea, but not to a greater extent than in a normal case. There was no appreciable change in the smaller type of cell in the case of Sydenham's chorea, in the specimen of which the subthalamic region was normal and no cortical alterations were noted. The vessels were all markedly congested, but not more than is usual in a patient dying of pneumonia or any other infectious disease. No thrombosed vessels were found.

The symptomatology of the corpus striatum is being slowly evolved, due in large measure to the work by S. A. K. Wilson, Mills, C. and O. Vogt, Hunt, Spiller, Marie and others. It is worth noting that our knowledge of the functions of the corpus striatum has been derived largely from clinicopathologic study, and while our presentation is of small moment, it may add a little to the sum total of our knowledge.

SUMMARY

1. A clinicopathologic study was made of a case of Sydenham's chorea and of one of Huntington's chorea.

2. In the typical case of Sydenham's chorea a mitral endocarditis was found. The pathology in the brain of acute cell changes with the marked congestion can be attributed to the acute infectious disease from which the patient died.

3. In the case of chronic chorea the process involved mainly the striatum (caudate and putamen) and the cortex; the changes being typical of a chronic degenerative process in these parts, selective in action.

4. No relationship could be found between the acute and chronic varieties of chorea.

5. The findings of epidemic encephalitis were not present in our case of Sydenham's chorea. When such are present, as in Marie's case, we believe them to be the result of the epidemic forms of encephalitis and not ordinary types of acute chorea.

TWO CASES OF BRAIN TUMOR WITH VENTRICULOGRAPHY

PETER BASSOE, M.D., AND CARL B. DAVIS, M.D.

CHICAGO

CASE 1.—*History.*—A boy, 10 years old, was admitted to the Presbyterian Hospital on June 10, 1922, complaining of headache, vomiting, failing vision and numbness in the extremities. The headache had commenced a year previously, was chiefly frontal and was often accompanied by vomiting. Impairment of vision had been progressing steadily since early in May. Numbness of the limbs, especially on the right side, had been present for two weeks. Double vision had been complained of a year before and lasted several months.

Examination.—There was distinct bulging of the forehead, and the superficial veins of the forehead and temples were prominent. In this region there was also tenderness on precussion. Hearing was normal. There was no paralysis of the extremities, and coordination was normal. The tendon reflexes were absent in the legs and present in the arms. The abdominal and plantar reflexes were normal. Sensation was normal. Dr. T. D. Allen reported: Bilateral papillitis, swelling of right disk being 2.5 diopters; of the left disk, 2 diopters; vision on the right, 20/50; on the left, less than 1/200. The fields of the right eye were generally contracted from 10 to 30 degrees; the upper nasal field was entirely gone. In the left eye, there was only a small island remaining in the upper temporal field close to the point of fixation; central vision was lost. In the right eye, there was total paralysis of the sixth nerve; nearly total paralysis of the external branches of the third, and slight paralysis of the fourth. In the left eye, there was total paralysis of the fourth nerve; slight paralysis of the sixth, and nearly total paralysis of the external branches of the third nerve. Ptosis and nystagmus were not present. The spinal fluid was under greatly increased pressure—58 mm. of mercury—was clear and pale yellow. The cell count was 8. The Ross-Jones globulin test gave a faintly positive reaction. There was also a slight Lange reaction (0112221000). The Wassermann test was negative with both blood and spinal fluid. The blood showed slight leukocytosis (10,700 to 13,200). Roentgenologic examination of the head showed a definite outward bulging in the left temporal region, unusually prominent suture lines, and a suggestion of digital impressions.

As there was considerable uncertainty as to whether the suspected tumor was in the posterior fossa or in the frontal region, it was decided to resort to ventriculography. On June 16, a needle was inserted into the posterior horn of the right lateral ventricle through a trephine opening. Fifty c.c. of fluid were withdrawn and 40 c.c. of air injected. Roentgenograms were taken with the head on either side and one with the forehead down. No air was seen in the picture obtained in the last named position. In the lateral views the ventricles were found to be rather large, but one of the anterior horns contained no air, and the anterior border of this ventricle appeared uneven. This appearance was obtained when the left side of the head was against the plate, and theoretically the right anterior horn should have been the occluded one. However, the roentgenologists, by comparing the two lateral plates, concluded that the left anterior horn was the one affected, and that

the air had not been given time to rise to the opposite side. The procedure was well borne by the patient, and a week later Dr. Allen found some decrease in the swelling of the disks and a little improvement in the vision of the left eye. On June 26, Dr. Davis turned down a large flap over the left frontal lobe, but no growth was found. On inserting a needle, 50 c.c. of fluid were removed from the left anterior horn, which we had thought was occluded. For two days the patient was in very poor condition. Dr. Allen found a further reduction of swelling of the disks on the day after the operation, and on June 29, the swelling of the left disk was not over half a diopter. There was also a wider range of ocular movements. On June 27, the leukocyte count was 30,000 but the temperature was only 99.4 F. No fever developed, but the patient gradually failed, and he died on July 8.

Necropsy Examination.—At necropsy the brain was found to be under very high pressure. The meningeal vessels were practically empty, the gyri very

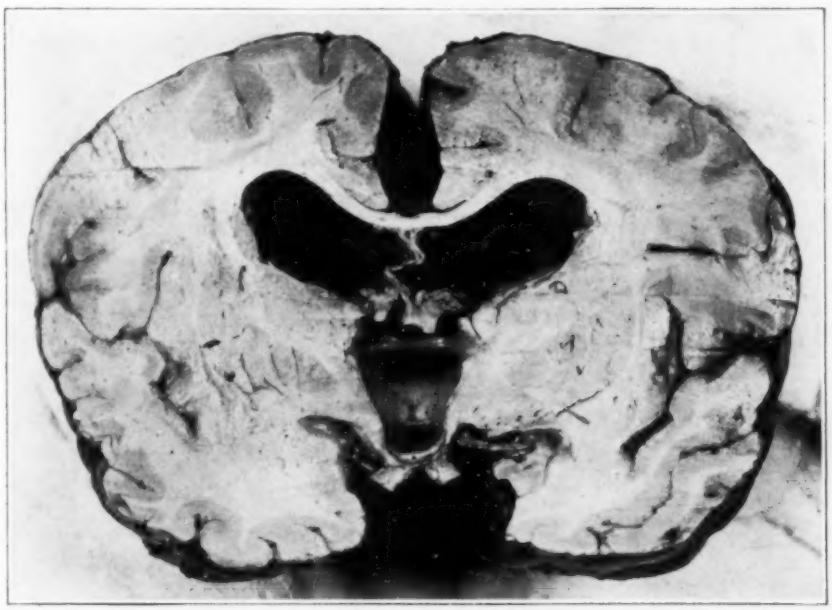

Fig. 1 (Case 1).—Symmetrical internal hydrocephalus.

flat, the sulci obliterated. A little pus was found in the left tympanum and mastoid.

After formaldehyd hardening and with empty ventricles the brain weighed 1,230 gm. On section, no change was found in the cerebrum except marked and perfectly symmetrical internal hydrocephalus. The enlargement of the third ventricle and aqueduct was especially marked (Fig. 1).

The under surface of both occipital lobes was raised like a dome and their gyri completely flattened by the upward pressure of the cerebellum. Externally the cerebellum showed no change except marked molding of the inferior portion at its line of contact with the foramen magnum.

On section, a large tumor mass was found to fill and greatly distend the upper part of the fourth ventricle (Fig. 2). The greatest dimensions of the tumor were 5 cm. from side to side and 3 cm. from above downward.

The lower part of the ventricle was not invaded. The upper portion of the tumor was dark reddish-black as a result of recent hemorrhage, while the lower portion was light gray. In its lower portion, the tumor appeared more firmly attached to the roof of the cavity than to its floor, while in the upper portion the opposite was true. The upper end of the tumor occupied the distended aqueduct of Sylvius and projected into the distended third ventricle.

Histologically, the tumor proved to be a glioma.

Case 2.—*History.*—A married woman, 34 years old, was admitted to the hospital July 27, 1922, on the service of Dr. T. D. Allen, to whom we are indebted for the privilege of studying and reporting this case. Seven years before she began to have epileptic attacks, preceding which she saw flashes of light. The convulsions were general and were sometimes associated with biting of the tongue and evacuation of the bladder. In the beginning the attacks occurred every day, and often several times a day, but they gradually decreased in frequency and severity. There was a little headache after the

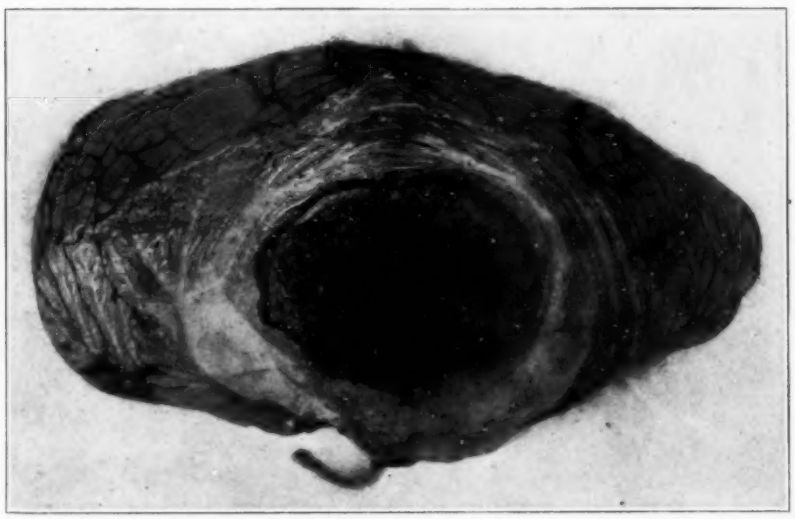

Fig. 2 (Case 1).—Tumor in upper part of fourth ventricle.

attacks, and sometimes at the beginning of the menstrual periods, but it never was severe, and in later years was infrequent. Vision began to fail about one year and seven months prior to admission, and during the last two months she had been practically blind, although some light perception was present part of the time. (As late as June 24, vision on the right was 20/100 and on the left 20/40.) This intermittence was very striking. At the time of admission, she began to complain of numbness and a pricking sensation in the right half of the face, including the mucous membranes of the nose, mouth and tongue. Formerly she had noticed a similar sensation in other parts of the body after the convulsions. Dr. Allen found that the pupils were small. The left pupil reacted normally, the right one somewhat sluggishly, to light. Bilateral papillitis was present with a swelling of 4 diopters of each disk. On examination, the tongue was found to protrude slightly to the left. The muscles of mastication were apparently weak on

both sides, but the patient did not complain of difficulty in chewing. There was no definite facial palsy, but the left eye was habitually wider open than the right. Hearing was good. Coordination was good. The tendon reflexes were practically normal; the left knee and ankle reflexes were slightly stronger than the right. The plantar reflexes were normal; the abdominal

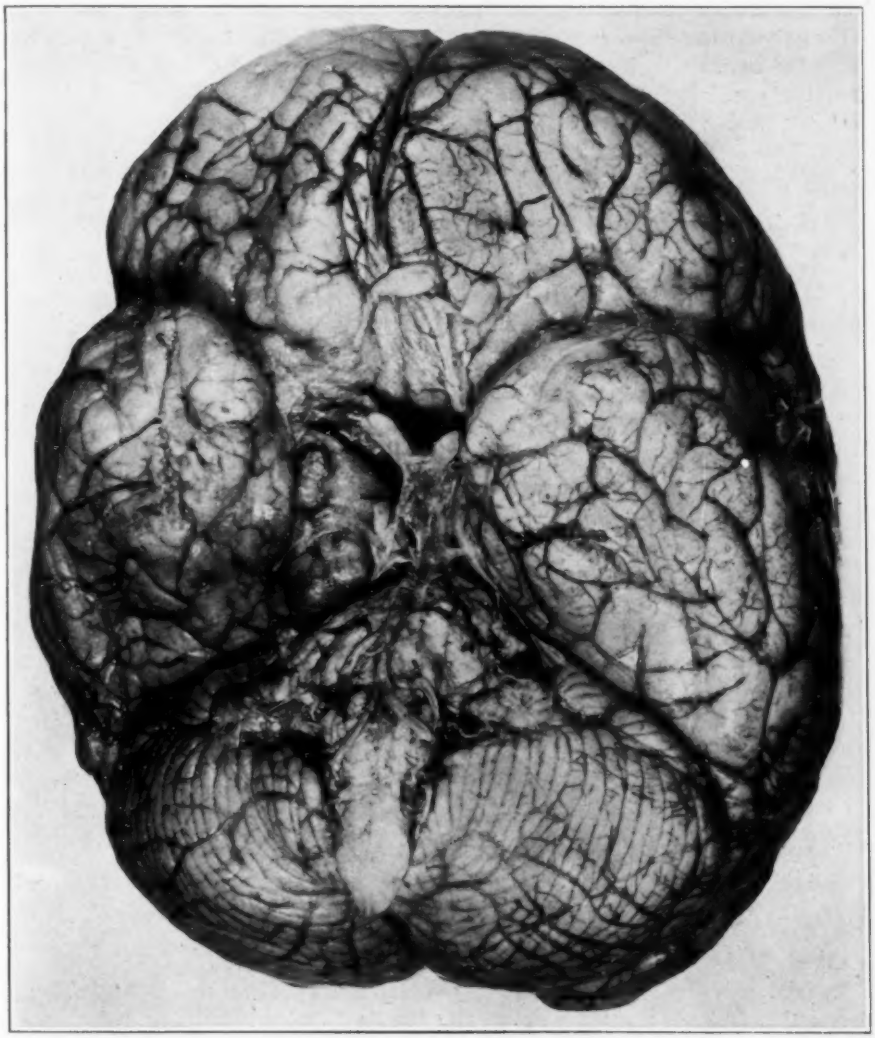

Fig. 3 (Case 2).—Base of brain showing tumor of right temporal and frontal lobes.

reflexes were diminished on the left. There was a questionable impairment of taste on the right half of the tongue and a very definite diminution of pain, touch and temperature sensation on the right side of the head, both on the face and posteriorly. There was less definite impairment of tactile and pain

sensation in all four extremities. The spinal fluid pressure was 12 mm. of mercury. All spinal fluid tests, as well as the blood Wassermann test, were negative. The leukocyte count was 13,400. On roentgenologic examination of the head no abnormality was noted, except that the posterior clinoid processes were indistinct. A tentative diagnosis was made of a tumor on the right side of the brain, so located as to make direct pressure on the chiasm, as it was not thought that the papillitis and blindness could be due to hydrocephalus or general increase in pressure. Although quite certain that the tumor was inaccessible, we yielded to the temptation of attempting to obtain a more definite localization by means of ventriculography. On August 15, Dr. Davis trephined and introduced air into the left lateral ventricle. Only about 20 c.c. of fluid was obtained, and possibly a slightly larger

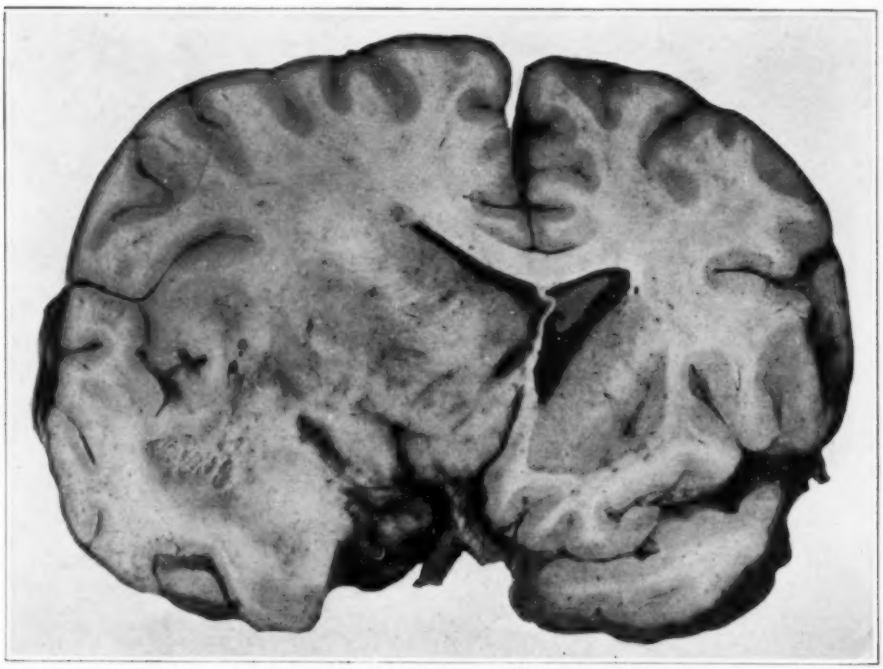

Fig. 4 (Case 2).—Cross section of tumor in temporal lobe.

amount of air was introduced. Films were taken with the head on either side, and with the forehead and occiput down against the plate. The description by the roentgenologist, Dr. Cassie B. Rose, omitting reference to the trephine holes, is as follows: "Film against the right side of the skull seems to show good filling of the lateral ventricle. Lateral view, with the left side against the film also shows the lateral ventricle except that the anterior horn is not made out. On anteroposterior view, there is on the left side a long, narrow shadow, probably a filling in the left ventricle, with a streak extending toward the right side. The right ventricle, however, is not made out. On postero-anterior view a decreased density is seen above the left orbit, probably the shadow of air in the left ventricle; right not made out."

On returning from the operating room, the patient, was somewhat cyanotic, but the pulse was regular and full. One hour later the pulse rate suddenly rose form 72 to 145, and the cyanosis increased. Twenty minutes later the patient died.

Necropsy Examination.—The dura was everywhere tense. The superior longitudinal sinus was empty and the brain squeezed tightly against the dura. The gyri were flat and the sulci largely obliterated.

The hardened brain weighed 1,475 gm. The right hemisphere appeared to be larger than the left, especially in the anterior portion. Looking at the inferior surface, we noted a grayish, rounded projection, firmer and slightly darker than the normal cortex, pressing against the anterior portion of the pons and displacing the chiasm toward the left (Fig. 3).

The fifth cranial nerve at its exit from the pons was in close contact with this growth. The right third nerve lay in a groove on the under surface of the projecting mass. The under surface of the right frontal lobe from a distance of 2 cm. in front of the chiasm also appeared darker than the surrounding brain tissue. The length of the whole tumor area along the posterior surface of the brain was 7 cm., its greatest width was 2 cm.

On section, the right temporal and frontal lobes were found to be occupied by an infiltrating, indistinctly-outlined, in places softened, tumor mass (Fig. 4). It invaded the cortex of the inferior surfaces and occupied roughly the lower two thirds of the right frontal lobe and the mesial two thirds of the temporal lobe. The ventricles on the right side were small, and, with the exception of the posterior horn and the end of the descending horn, were nearly obliterated. The lateral ventricle on the left side was not distended, and its widest point was seen at the cross-section illustrated in Figure 4.

In the occipital lobe, a few small hemorrhages marked the path of the ventricular puncture.

The cerebellum showed no gross change, except considerable molding at its contact with the foramen magnum.

Histologically, the tumor proved to be a glioma.

COMMENT

We report these cases chiefly to point out that in the hands of novices like ourselves ventriculography is likely to be misleading and dangerous. In the first case, sufficient air had not been introduced into the posterior horn to fill the anterior horns completely. In the second case, in which the ventricles were unexpectedly small, we evidently introduced too much air, and death was caused by the acute compression. Aside from determining the absence of hydrocephalus, we did not learn to locate the tumor any more accurately than we had by the neurologic symptoms. However, we are willing to admit that in selected cases and in hands more skilful than ours the method occasionally will lead to a correct localization and successful removal of a tumor which cannot be located by other known methods.

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THE DIFFERENCE BETWEEN A MUSCULAR AND A NEUROMUSCULAR INTERPRETATION OF WALKING*

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NEW YORK

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I. Introduction

Walking is a highly complicated process. The underlying mechanisms, in view of their importance in all studies of motility, have attracted attention at least since the days of the Weber brothers, nearly one hundred years ago. The general conception of the movements of the four extremities has not changed essentially since it was first described. These movements were recently described thus: "The right upper and left lower limbs are coupled together and swing forwards or backwards at the same time, flexion of one arm occurring with extension of the leg on the same side." [1] In other words, both the homolateral limbs as well as the pairs of arms and legs, respectively, move in opposite directions. This conception is based on ordinary observations and on the action of certain definite muscles. Though it has been recognized since the days of Huxley that the front of the leg is, in reality, the dorsal surface and though this has been abundantly proved by various anatomists who have studied the comparative anatomy and embryology of the limbs, the significance of this has apparently attracted little attention among neurologists and has not been used in the interpretation of group movements, normal or abnormal, of man. The process of walking has therefore been discussed from the point of view of muscular action, on one hand, and

* Read by title at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

1. Riddoch, George, and Buzzard, E. Farquhar: Reflex Movements and Postural Reactions in Quadriplegia and Hemiplegia, with Especial Reference to Those of the Upper Limb, Brain **44**:462, 1921.

from the point of view of its general control as a reflex mechanism by the spinal cord on the other. The classical work of Sherrington has established definite laws for this reflex action and has also indicated which muscles are involved in certain phases of reflex stepping (Fig. 1, Table 1). However, in all this, no attempt seems to have been made to interpret walking from a point of view which considers the embryology and evolutionary origin of the muscles involved. I shall attempt to show that the spinal cord controls, by its integrations, large groups of muscles which are related, not only embryologically, but also by virtue of their common nerve supply. On this basis it may be shown that the integrations controlling walking are among the foundations of the nervous system and constitute primitive motility patterns. All

TABLE 1.—The Muscles Shown in Figure 1 Arranged in Ventral and Dorsal Groups *

	A		B	
	Ventral	Dorsal	Ventral	Dorsal
I.	.....	I. Psoas M. Gluteus minimus R. Rectus femoris F. Tensor fasciae femoris brevis S. Sartorius lateralis S'. Sartorius medialis	0. Quadratus femoris 3. Adductor minor 4. Adductor major (a part) 5. Semimembranosus 6. Biceps femoris posterior	
II.	B. Biceps femoris posterior G. Gracilis T. Semitendinosus	.....	.....	1. Crureus 2. Vasti
III.	.....	A. Tibialis anticus L. Extensor longus digitorum P. Peroneus longus E. Extensor digitorum brevis	7. Gastrocnemius 8. Soleus 9. Flexor longus digitorum	

* The first group in both tables includes muscles acting at the hip, the second those acting at the knee, the third those acting at the ankle.

the neurons engaged in producing patterns necessary to gait are of spinal origin. On these are superimposed other patterns appearing later in the course of development and more complicated mechanically.

In order to appreciate the significance of the various integrations controlling motility, it is necessary first to understand the most primitive of these and the manner in which they control muscles. Emerging from this method of interpretation of movement—which has been designated the neuromuscular [2] and which considers the grouping of the peripheral motor neurons and their end-organs, the muscles—the idea

2. Kraus, Walter M.: A Principle Hitherto Undescribed of the Physiology of Movement and Posture. The Primitive Integration of Movement in Vertebrates, Arch. Neurol. & Psychiat. **7**:381 (March) 1922.

will be developed that though the actual visible movements involved in walking indicate that in reality the homolateral limbs move in opposite directions, when their control by the nervous system is considered, these opposite movements are equivalent to movement in the same direction. Furthermore, it will be shown that there exist corresponding opposite phases of gait in which the analogues of the activity of singly hinged appendages in primitive animals can be made out, and

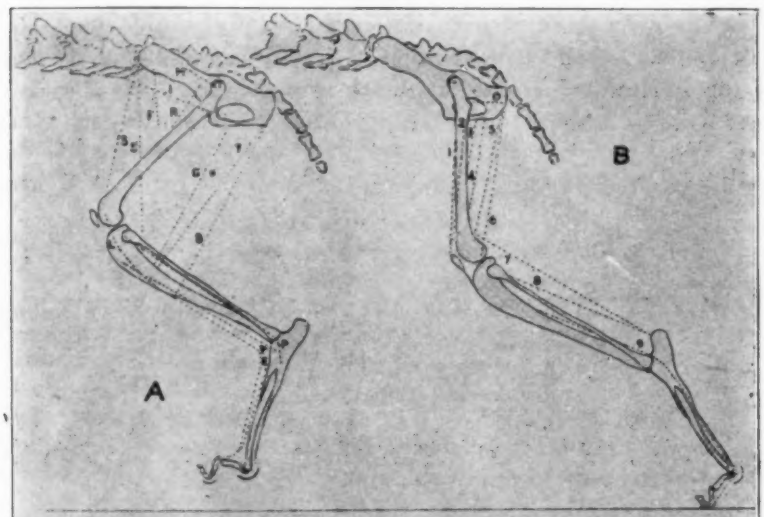

FIG. 5.

Figure illustrating the muscles actually observed by experimental analysis to be engaged in contracting in the flexion-phase A and in the extension phase B of the reflex step of the cat.

A.	*B.*
A, tibialis anticus.	0, quadratus femoris.
B, biceps femoris posterior.	1, crureus.
E, extensor brevis digitorum.	2, vasti.
F, tensor fasciæ femoris brevis.	3, adductor minor.
G, gracilis.	4, adductor major (a part).
I, psoas.	5, semimembranosus.
L, extensor longus digitorum.	6, biceps femoris posterior.
M, gluteus minimus.	7, gastrocnemius.
P, peroneus longus.	8, soleus.
R, rectus femoris.	9, flexor longus digitorum.
S, sartorius lateralis.	
S', sartorius medialis.	
T, semitendinosus.	

Fig. 1.—Reproduced from *Brain*, volume 33, page 22, 1910, from the article by C. S. Sherrington on "Remarks on the Reflex Mechanisms of the Step."

that superimposed on these is another pair of corresponding opposites also controlled by the spinal cord and based on the development, in the course of evolution, of the three-hinged appendages of the land-living vertebrate. Such an analysis of the control of walking by spinal integrations demands an entirely new point of view toward posture and progression patterns, not only in normal man but also in all disorders of the central nervous system in which these neurons are disturbed.

II. THE ANATOMIC AND PHYSIOLOGIC BASES OF THE NEUROMUSCULAR MECHANISM

As has been said before,[2] movements are now described in such terms as extension, abduction, internal rotation, etc. In other words, we describe the activity of a neuromuscular mechanism in terms of the muscular part of that mechanism alone, disregarding the neurologic element of movement and its control by the spinal cord and its peripheral nerves. The neural part of the mechanism is represented by peripheral motor neurons. The activation of groups of these neurons in various ways by the spinal cord is carried out by neurons within the spinal cord and is known as integration. When we describe in terms of muscular action only, complicated movements guided by integrations of the nervous system at levels limited to or higher than the spinal cord,

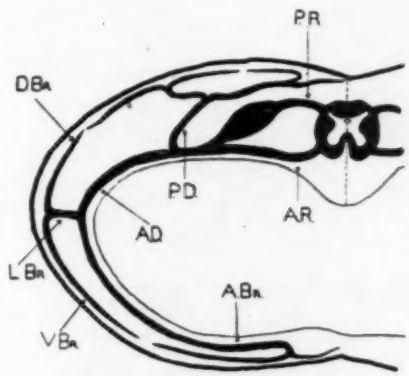

Fig. 2.—Adapted from "Human Anatomy" by G. A. Piersol, Part 2, p. 1284, Fig. 1085. *D. Br.*, dorsal branch; *V. Br.*, ventral branch; *L. Br.*, lateral branch; *A. Br.*, anterior branch; *P. R.*, posterior root; *A. R.*, anterior root; *P. D.*, posterior division; *A. D.*, anterior division.

we omit consideration of the spinal patterns and control of the final neuromuscular mechanism.

The nervous system is unquestionably built up of a series of physiologic levels of which the higher or more recently developed, phylogenetically speaking, carry out functions which are different from the lower. As Herbert Spencer remarked, "Integration keeps pace with differentiation." As animals become more and more differentiated, more and more needful of performing new kinds of movements in pattern, integrations appear to carry out these group movements. It is my opinion that we must have some conception of the integrating action of the lowest levels in order to interpret the higher. We must know how the spinal cord integrates movements in order to understand the activities and patterns of higher integrations superimposed in the course of

evolution and ultimately to understand why it has become necessary for these higher integrations to appear at all. It is necessary to consider in detail the general plan of the peripheral nerves, the general plan of the innervation of muscles by them and the embryologic grouping of the muscles themselves in order properly to examine integration of movement by the spinal cord.

A typical thoracic spinal nerve having nothing to do with the innervation of limbs shows a number of divisions after union of the anterior and posterior spinal roots. The trunk so formed divides into an anterior and posterior division. The anterior division divides further into a lateral and an anterior branch, the lateral branch dividing into a dorsal

TABLE 2.—Division of the Nerves of the Extremities into Dorsal and Ventral Groups

Origin	Groups	Nerves
	Upper Extermity	
Brachial plexus	Dorsal trunks (posterior cord)	Dorsal scapular Long thoracic Suprascapular Subscapular (2) Thoracodorsal Axillary Radial
	Ventral trunks (lateral and medial cords)	Nerve to subclavius Anterior thoracic (2) Musculocutaneous Median Ulnar
		Lower Extremity
Lumbosacral plexus	Dorsal trunks	Superior gluteal Inferior gluteal Nerve to piriformis Femoral Peroneal
	Ventral trunks	Obturator Nerve to obturator internus and superior gemellus Nerve to quadratus femoris and inferior gemellus Tibial

and a ventral (Fig. 2). As far as the axial musculature is concerned, the portions of it developed from the ventrolateral portions of the embryo are supplied by the anterior division and its branches, while the dorsal musculature is supplied by the posterior branches. Thus, a simple scheme results from a physiologic point of view. There emerges the notion that, in general, movements of extension are guided by the posterior division, while flexion is guided by the anterior division. The lateral and rotatory movements produced by these groups of muscles are due to overactivity of the right and left halves of either the dorsal or the ventral groups or both. One theory assumes that the limb plexuses are made up only of the lateral branch of this division. For the purposes of this discussion, it is not important to try to reach a decision as to which of these theories is correct.

The primitive extremities of the embryo are flattened buds having a dorsal and ventral surface. The "premuscle mass" on the dorsal side gives rise to muscles the majority of which, in the adult, have an extensor function. The "premuscle mass" on the ventral side gives rise to muscles the majority of which have a flexor function. The

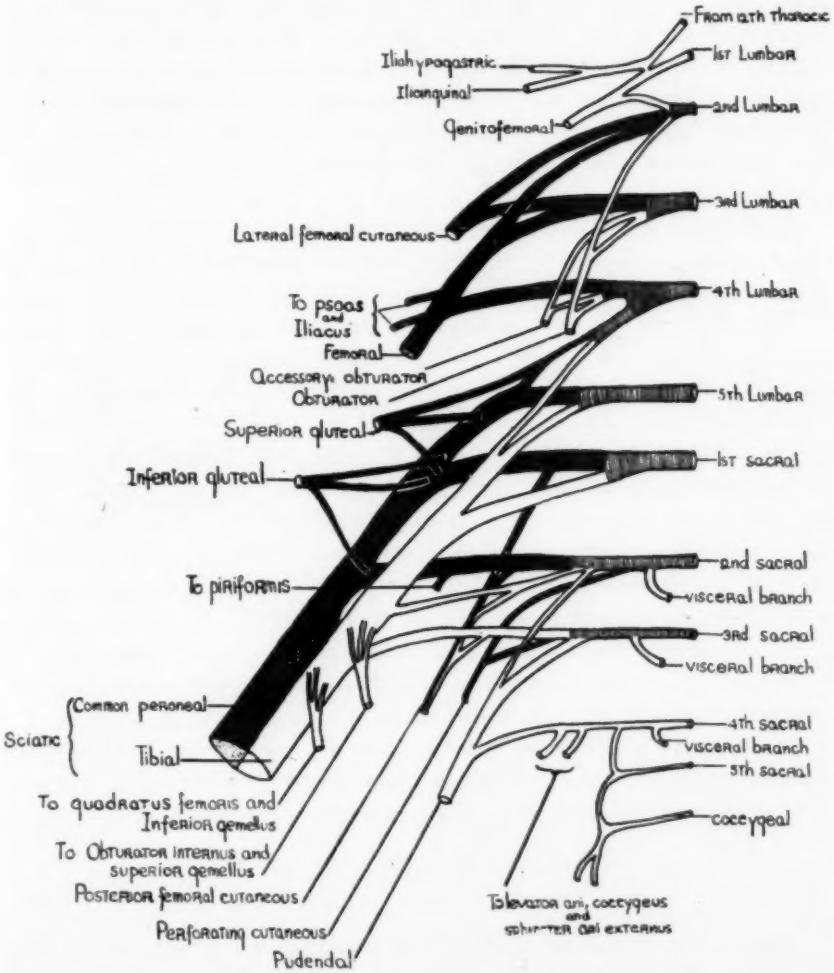

Fig 3.—Plan of the lumbar, sacral and pudendal plexuses. Black, dorsal; white, ventral; cross-hatched, combined. (Modified from Gray's "Anatomy," pp. 949 and 958).

crux of the whole situation lies in the fact that certain muscles have a function opposite to that which would be expected from a knowledge of the side of the limb bud (ventral or dorsal) from which they develop. For example, the iliopsoas group develops from the dorsal

muscle mass of the lower limb bud, but has a ventral function, that is, flexion.[2] This is an old and well-known fact, and is due to rotation of the leg in the course of evolution.

To return now to the question of innervation, we find that the embryology of the muscles is beautifully emphasized by their innervation. It is well known that the nerves of the brachial and lumbosacral plexuses divide into two definite groups, one of which is posterior or dorsal and the other anterior or ventral. In the arm, the ventral branches of the brachial plexus innervate the subclavius and pectoral muscles as well as all the muscles supplied by the musculocutaneous, median and ulnar nerves. The remaining nerves are dorsal (Table 2). The same division in the lumbosacral plexus is indicated by Figure 3 and Table 2.

A detailed table of muscles based on their derivation from the dorsal or ventral aspects of the limb bud and their supply by the dorsal or ventral branches of the limb plexuses shows that those muscles which develop from the dorsal layers of the bud are supplied by the dorsal branches of the limb plexus and those which develop from the ventral layers by the ventral branches. The analogy to the condition in the axial musculature is obvious (Tables 2, 3, 4 and 5).[3]

From all this we arrive at some possibility of schematization. In the nerves and muscles of the upper and lower limbs we find, as in the case of the iliopsoas mentioned before, that muscles which would be expected to have an extensor action have in reality a flexor action and vice versa. In other words, were we to group the muscles acting in any given movement merely on the basis of their activity as muscles and leave out of consideration their embryology and activating nerves, we would be leaving out entirely the division of these nerves and of their end-organs, the muscles, into large ventral and dorsal groups.

To illustrate how this information may be used for discovering just how the spinal cord may integrate movement, let us consider the use of relatively simple spinal integrations in the process of walking.

III. TECHNIC USED IN ANALYZING THE PROCESS OF WALKING

Simple observation of walking and running in a normal man does not permit of a precise analysis of the various phases of gait. The movements are too rapid and the transitions between flexion and extension, etc., are too small to give accurate results. Advantage was there-

3. Lewandowsky, M.: Handbuch der Neurologie. Flatau, Edward: Die Motorische, Sensible und Reflex Segmentierung im Rückenmark, Allg. Neurol. **1**, Pt. 2, p. 659, 1910. Cunningham, D. J.: Textbook of Anatomy, New York, William Wood & Co., 1918, p. 743. Braus, Hermann: Anatomie des Menschen, Bewegungsapparat, Berlin **1**: 1921.

fore taken of cinematographic pictures by which a considerable number of successive phases of walking and running could be obtained.

The pictures taken were a series from *X* to *Z* (Fig. 4). *X Y* and *Z* were indicated on a black drop by sheets of white paper. *Y* indicates

TABLE 3.—Division of the Muscles of the Upper Extremity into Dorsal and Ventral Groups

	Dorsal		Ventral
1	Levator scapulae	1	
2	Serratus anterior	2	
3	Rhomboideus major	3	
4	Rhomboideus minor	4	
5	Supraspinatus	5	
6	Infraspinatus	6	
7	Teres minor	7	
8	Deltoid	8	
9	Subscapularis	9	
10	Teres major	10	
11	Latissimus dorsi	11	
12		12	Subclavius
13		13	Pectoralis major
14		14	Pectoralis minor
15		15	Biceps brachii
16		16	Brachialis *
17		17	Coracobrachialis
18	Brachioradialis	18	
19	Extensor carpi radialis longus	19	
20	Extensor carpi radialis brevis	20	
21	Supinator	21	
22	Extensor pollicis longus	22	
23	Extensor indicis proprius	23	
24	Abductor pollicis longus	24	
25	Extensor pollicis brevis	25	
26	Extensor communis digitorum	26	
27	Extensor carpi ulnaris	27	
28	Extensor minimi digiti quinti	28	
29	Anconeus	29	
30	Triceps	30	
31		31	Pronator teres
32		32	Flexor carpi radialis
33		33	Palmaris longus
34		34	Flexor digitorum sublimis
35		35	Flexor digitorum profundus
36		36	Flexor pollicis longus
37		37	Pronator quadratus
38		38	Lumbricalis 1
39		39	Lumbricalis 2
40		40	Lumbricalis 3
41		41	Lumbricalis 4
42		42	Abductor pollicis brevis
43		43	Opponens pollicis
44		44	Flexor pollicis brevis (lateral head)
45		45	Flexor pollicis brevis (medial head)
46		46	Flexor carpi ulnaris
47		47	Adductor pollicis obliquus
48		48	Adductor pollicis transversus
49		49	Interosseus volaris 1
50		50	Interosseus volaris 2
51		51	Interosseus volaris 3
52		52	Interosseus dorsalis 1
53		53	Interosseus dorsalis 2
54		54	Interosseus dorsalis 3
55		55	Interosseus dorsalis 4
56		56	Opponens digiti quinti
57		57	Flexor digiti quinti brevis
58		58	Abductor digiti quinti

* Supplied by both dorsal and ventral nerves.

the point at which the subject was directly in front of the camera and *X* and *Z* two points equidistant from *Y* on either side. The distance *X*—*Z* is equal to 17 feet. In order to include positions *X* and *Z*, a panoramic series was used. Of the series reproduced, seventeen pictures were

TABLE 4.—Division of the Muscles of the Lower Extremity into Dorsal and Ventral Groups

	Dorsal		Ventral
1	Iliacus	1	
2	Psoas major	2	
3	Psoas minor	3	
4	Pectineus *	4	
5	Sartorius	5	
6	Rectus femoris	6	
7	Vastus lateralis	7	
8	Vastus medialis	8	
9	Vastus intermedius	9	
10	Tensor fasciae latae	10	
11	Gluteus minimus	11	
12	Gluteus medius	12	
13	Piriformis	13	
14	Gluteus maximus	14	
15	Biceps femoris, short head	15	
16		16	Biceps femoris, long head
17		17	Semitendinosus
18		18	Semimembranosus
19		19	Adductor magnus
20		20	Obturator externus
21		21	Adductor longus
22		22	Adductor brevis
23		23	Gracilis
24		24	Obturator internus
25		25	Superior gemellus
26		26	Interior gemellus
27		27	Quadratus femoris
28	Tibialis anterior	28	
29	Extensor hallucis longus	29	
30	Extensor digitorum longus	30	
31	Peroneus tertius	31	
32	Peroneus longus	32	
33	Peroneus brevis	33	
34	Extensor digitorum brevis	34	
35		35	Gastrocnemius
36		36	Soleus
37		37	Plantaris
38		38	Popliteus
39		39	Tibialis posterior
40		40	Flexor digitorum longus
41		41	Flexor hallucis longus
42		42	Quadratus plantae
43		43	Abductor digiti quinti
44		44	Flexor digiti quinti brevis
45		45	Opponens digiti quinti
46		46	Plantar interosseus 1
47		47	Plantar interosseus 2
48		48	Plantar interosseus 3
49		49	Dorsal interosseus 1
50		50	Dorsal interosseus 2
51		51	Dorsal interosseus 3
52		52	Dorsal interosseus 4
53		53	Adductor hallucis
54		54	Lumbricalis 1
55		55	Lumbricalis 2
56		56	Lumbricalis 3
57		57	Lumbricalis 4
58		58	Abductor hallucis
59		59	Flexor digitorum brevis
60		60	Flexor hallucis brevis

* Supplied by both dorsal and ventral nerves.

used for analysis. It required three and one-fifth seconds to walk from *X* to *Z* and about one second to walk one pace. By a stroke of good luck the picture showing one leg in support and the other in maximum flexion (No. 25) was but one from *Y*, that is, almost directly in front of the camera (Figs. 4 and 5). The degree of rotation of the body appearing at *X* and *Z* is shown in Figure 6. This may be compared to Figure 5.

TABLE 5.—Division of the Muscles of the Axis into Dorsal and Ventral Groups

	Ventral	Dorsal
Muscles of the Back:		
Serratus posterior superior		+
Serratus posterior inferior		+
Splenius		+
Sacrospinalis		+
Iliocostalis		+
Longissimus		+
Spinalis dorsi		+
Semispinalis		+
Multifidus		+
Obliquus capitis inferior		+
Obliquus capitis superior		+
Rectus capitis posterior major		+
Rectus capitis posterior minor		+
Rotatores		+
Interspinales		+
Intertransversarii	+	
Trapezius		+
Muscles of the Neck:		
Scalenus anterior	+	
Scalenus medius	+	
Scalenus posterior	+	
Longus capitis	+	
Rectus capitis anterior	+	
Longus colli	+	
Rectus capitis lateralis	+	
Sterno-kleido-mastoid	+	
Muscles of the Thorax:		
Intercostales	+	
Levatores costarum	+	
Subcostales	+	
Transversus thoracis	+	
Muscles of the Abdominal Wall:		
Obliquus externus abdominis	+	
Obliquus internus abdominis	+	
Cremaster	+	
Transversus abdominis	+	
Pyramidalis abdominis	+	
Rectus abdominis	+	
Quadratus lumborum	+	
Muscles of the Perineum:		
Sphincter ani externus	+	
Corrugator cutis ani	+	
Transversus perinei superficialis	+	
Bulbocavernosus	+	
Ischiocavernosus	+	
Sphincter urethrae membranaceae	+	
Transversus perinei profundus	+	
Muscles of the Pelvis:		
Levator ani	+	
Coccygeus	+	

Except for careful mathematical analysis, this rotation is not important, though in all analyses it should be regarded as a source of error in drawing conclusions. Before taking the pictures it was determined that the difference between having the subject walk part of the circumference of a circle indicated by *X, W, Z* (Fig. 4), and walking a straight line, *X, Y, Z*, was so slight as to make its carrying out unimportant. This difference, the distance from *Y* to *W*, is only a foot and a half.

A series of cinematographic pictures was projected one after another on a wall at a distance of 12 feet. The images produced were 19 inches high. The method of determining the successive movements or changes in the angular relations of the thigh and torso, or of the knee and the thigh, was to trace a series of pictures with, in the first instance, the torso of each successive picture superimposed on the preceding one. The thigh movements are thus made out (Figs. 7 and 8). In tracing the thigh-knee relations, the thigh was taken as a constant

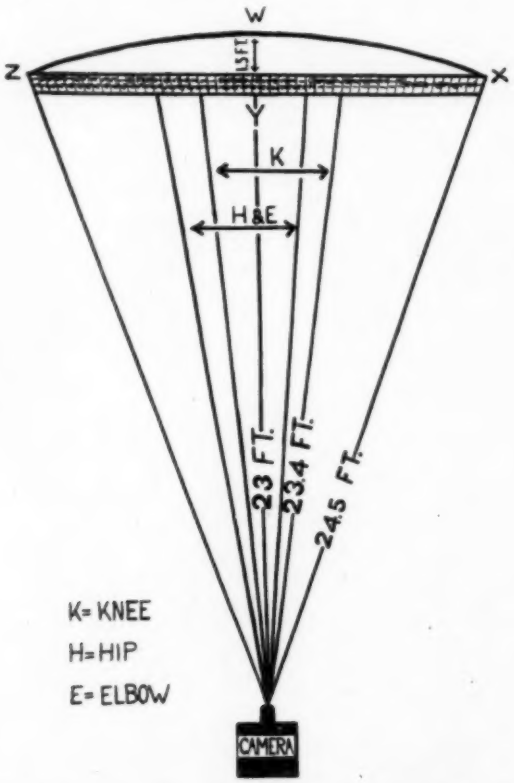

Fig. 4.—Diagram representing the distance relations of the patient to the camera. The patient walked a straight line from *X* to *Z*. The small numbers between *X* and *Z* each represent a picture. For further explanation see text, section III.

(Figs. 9 and 10). The elbow movements were determined by the same method (Fig. 11). Because of rotation, the shoulder movements cannot be illustrated easily and have been omitted. Certain precautions must be taken in such successive tracings in which changes in the positions of muscles cause changes in contour. The difference in the lower posterior thigh line, caused by changing positions of the hamstrings,

is illustrated in Figure 12, in that *A* shows the contour in flexion at the knee, *B* the contour in extension at the knee. In all drawings the anterior thigh line was used as the standard.

The horizontal has been inserted in all the pictures of the torso-thigh series. In the thigh-knee series the horizontal for the first tracing of each series has been inserted. A vertical line through the central position between ventral and dorsal and at right angles to the floor line was put into the torso-thigh cuts to indicate tilting of the torso and the relative change of the center of gravity in these positions.

It is obvious that a complete pace must be taken for analysis. It so happened that seventeen pictures were needed to show this. If,

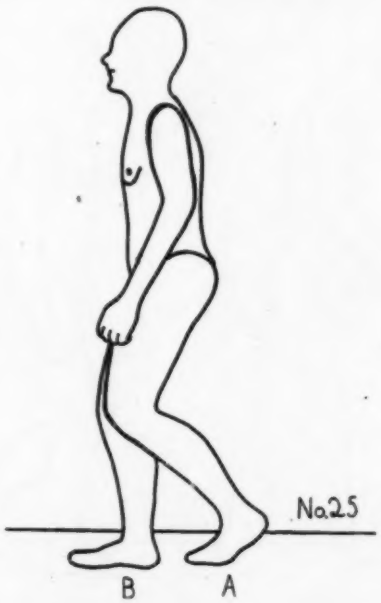

Fig. 5.—This tracing shows the left leg, A, in the maximum position of the flexion reflex and the right leg, *B*, in the maximum position of support. These constitute the third and fourth phases of walking. The actual picture, as well as the positions, are mathematically midway between those shown in Figures 13 and 14. The thighs are seen to be in alinement with one another. Flexion at the knee of the right leg, *B*, is apparent.

therefore, the first and seventeenth are the same, the ninth is the middle picture and must represent a stage midway between, and one in which the legs are in mirrored positions of those in the first and seventeenth. Furthermore, it would be expected that midway between the first and ninth and the ninth and seventeenth there would occur the maximum position of flexion reflex in one leg and a maximum position of support in the other. The analysis bore this out, and the fifth and thirteenth

represented these. To check this, they were compared. The arm positions were also used to determine the accuracy of the conclusions. When the arms are parallel to one another, one leg is in maximum support, *B*, and the other in maximum flexion, *A* (Fig. 5). An analysis of succeeding pictures furnishes a check to the selection of the various stages of the pace, irrespective of any suppositions on purely mathematical grounds. Still another method of determining by analysis of the knee movements that the fifth and thirteenth are indeed maximum positions of flexion reflex and support will be illustrated below. The actual pictures used were numbers 21 to 37 for the hip, shoulder and elbow and numbers 17 to 33 for the knee. A slightly

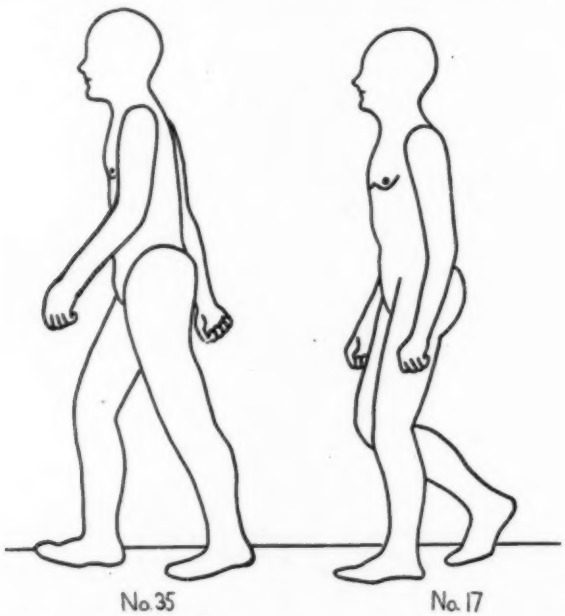

Fig. 6.—Rotation produced in a paroramic series of pictures, due to the patient's walking in a straight line. To avoid this rotation the patient should walk along the arc of a circle (Fig. 4, *X*, *W*, *Z*).

different series was taken for the knee in order to emphasize the difference in the extent of oscillation during the posture and flexion phases.

IV. MOVEMENTS DURING WALKING

For the purposes of the present analysis of the activity of the legs in walking, the assumption has been made that it is composed of four simple activities, namely: (1) the ventral, finlike movement of the lower limbs at the hip; (2) the dorsal, finlike movement of the lower limbs at the hip; (3) the flexion reflex element, and (4) the support element.

Finlike Movement at the Hip.—For the moment let us omit the last two elements. If the assumption concerning these four activities be true, one may expect that in the process of walking the leg undergoes a pendulum-like action from the points of attachment of the leg at the transervse plane of the hip. Such an action was postulated in 1836 by the brothers Weber, though they attributed this activity not to muscular but to gravity effects. Both Vierordt and Duchenne of Boulogne have considered that this activity is a part of the process of walking, carried out not by gravity, but by muscles. In an analysis of a complete pace of a normal person by means of tracings of successive cinematographic pictures, it has been found that in the process of walking the movement at the hip is continuous (Figs. 7 and 8).

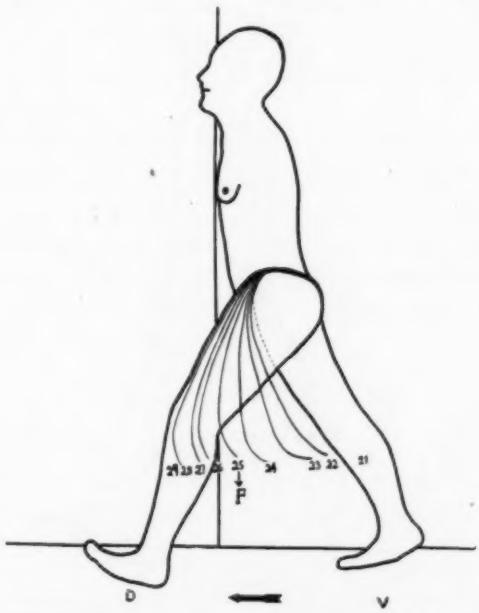

Fig. 7.—Consecutive tracings of the movement of the thigh in its progress from the ventral to the dorsal position. Compare with Figure 8.

It is well recognized now that the singly-hinged appendages of the fish preceded the three-jointed appendages of land vertebrates, such as amphibia. The most primitive grouping of movements by the spinal cord in the process of progression must have been one which correlated the activities of muscles much simpler than our own which lay on the ventral and dorsal aspects of the appendages, respectively. It seems obvious that the two groups of muscles which carried the fins in opposite directions must be controlled by spinal neurons permitting reciprocal innervation, so that when an agonist contracts its antagonist

is relatively relaxed. In order to realize this primitive finlike movement of a singly hinged appendage in man, it is necessary to regard the human leg as a rod and to ascertain whether there is a stage in the process of walking at which it assumes either a forward (dorsal) or backward (ventral) position. It is also necessary to determine whether these positions occur at regular intervals and whether they appear in definite relation to the two other elements of gait described by Sherrington as the extensor and flexor phases (Fig. 1).

It has been found that these two ventral and dorsal positions of either leg regarded as a rod do occur at regular intervals in the course of walking (Figs. 13 and 14), and it is found, furthermore, that

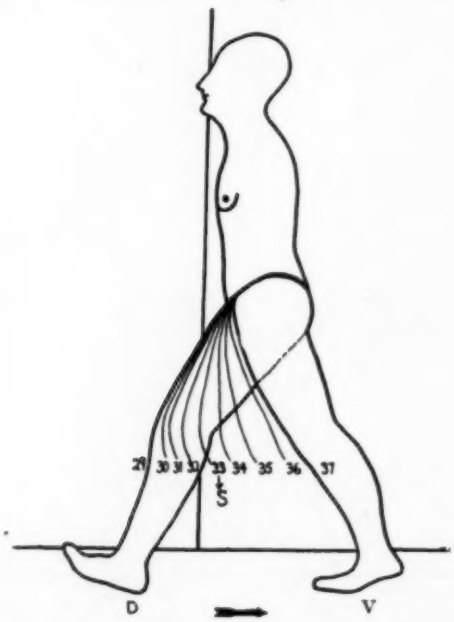

Fig. 8.—Consecutive tracings of the movement of the thigh in its progress from the dorsal to the ventral position. Compare with Figure 7.

midway between the appearance of the ventral and dorsal positions the flexion reflex element occurs, while the support element occurs midway between the appearance of the dorsal and ventral positions. It seems apparent, therefore, that in the process of walking the two primitive finlike movements at the hips can be made out with ease. That they occur rhythmically, that they are diphasic, continuous and represent alternating movements of the dorsal and ventral groups of hip muscles is illustrated by Figures 7 and 8. The rodlike positions are seen only at two stages of gait and are concealed at all other stages, due to the addition of the support and flexion elements.

The Flexion Reflex Element.—Since these movements which have just been described are necessarily more primitive than those of the three-jointed limbs of the land animal, it is assumed that the elements in walking developed by the land animal are considerably more complex and do not represent simple pendulum-like activities. As a matter of fact, the flexion reflex element represents an alternation of movement at the three great joints. This is beautifully demonstrated by an examination of the muscles given by Sherrington [4] in his paper, "Remarks on the Reflex Mechanism of the Step," as those causing the extensor and flexor phases of gait. What Sherrington called the flexion phase of reflex stepping corresponds precisely to the third element of gait in this analysis. Figure 1 and Table 1, taken from Sherrington's paper, illustrate this. The table of muscles, to which there has been added the words "ventral" and "dorsal," indicates the alternation of movement—dorsal at the hip, ventral at the knee and dorsal at the ankle.

The Support Element.—The extensor phase which Sherrington described shows precisely the opposite picture. That omission of the neuromuscular mechanism may lead to an attempt physiologically to group movements obviously not susceptible to such grouping is by now apparent. Two examples of such an attempt appear in Walshe's [5] paper on "The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs." He says:

> I have said above that all the flexors of the limb are activated in the flexion reflex. These include hip and knee flexors and the dorsiflexors of the ankle and digits. Now, of these, the dorsiflexors, in anatomical nomenclature, are named extensors. There can be no doubt, however, that these muscles must be grouped physiologically with the flexors. . . . In view of the fact that the "extensor response" is a part of a general movement of flexion while the toe movement in the normal plantar response, the "flexor response," is, physiologically considered, a movement of extension, it is unfortunate that the accepted terminology is the direct converse of the facts. The expressions "extensor response" and "flexor response" are widely used by neurologists in this country, and I have therefore been compelled to make use of them, though it is very difficult to prevent confusion in the mind of the reader when speaking of a "crossed extension reflex" and of a "crossed extensor response"; two very different phenomena involving antagonistic groups of muscles. I wish, therefore, to emphasize that these empirical and misleading names are used here in their accepted sense, and have no physiological connotation such as the terms "flexion reflex" and "crossed extension reflex" have. I have consequently placed them in inverted commas to avoid as far as possible all confusion.

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4. Sherrington, C. S.: Remarks on the Reflex Mechanism of the Step, Brain **33**:22, 1910-1911.

5. Walshe, F. M. R.: The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, Brain **37**:269, 1914-1915.

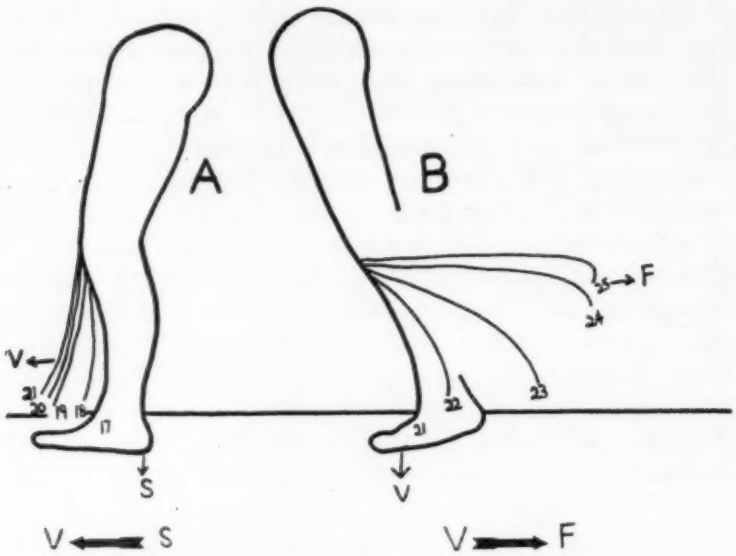

Figure 9

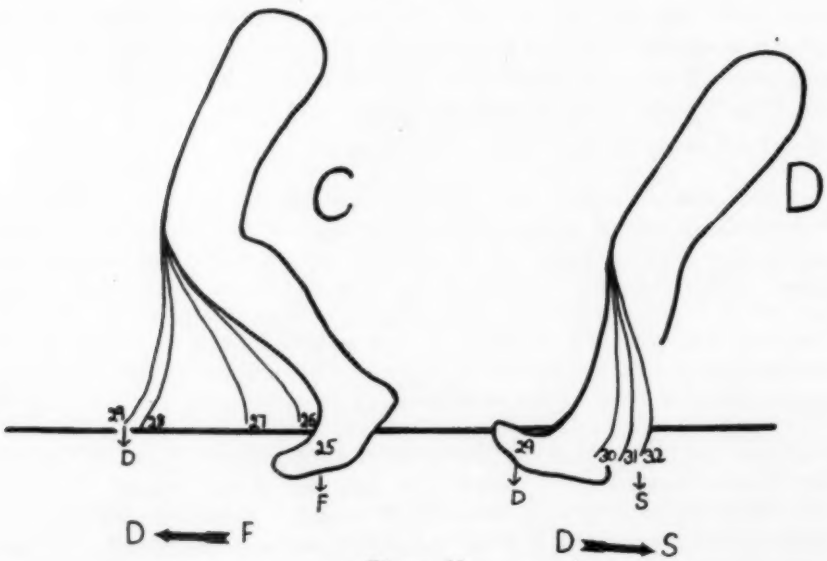

Figure 10

Figs. 9 and 10.—These illustrate the tetraphasic character of the knee movements. *A* and *D* constitute the diphasic posture element, *B* and *C* the diphasic flexion element. Only four tracings are shown in *D* as the next picture showed a position in the knee slightly in advance of its predecessor. This indicates a slight difference in the speed of walking or in the speed of taking the pictures. As a result only four pictures instead of five are needed to represent the last phase of posture in this particular pace. *A*, second phase of the support element; *B*, first phase of the flexion element; *C*, second phase of the flexion element; *D*, first phase of the support element.

Compare this statement with the data of Figures 1 and Table 1.

This illustrates how misleading and confusing grouping of muscle movements, as such and as they appear in certain reflexes, may become. The following quotation from Mackenzie [6] gives a different and sounder point of view, but one which leads to confusion when applied to Walshe's terms.

> The front of the leg and dorsum of the foot belong morphologically to the general extensor surface of the limb, corresponding with the back of the forearm and the dorsum of the hand. The back of the leg and sole of the foot correspond with the front of the forearm and palm of the hand, so that what we call flexion is really overextension. It is usual, however, and it will be followed here, to use the term flexion for the movement by which the dorsum of the foot is bent toward the front of the leg; the extension for the movement by which the sole of the foot is directed toward the back of the leg.

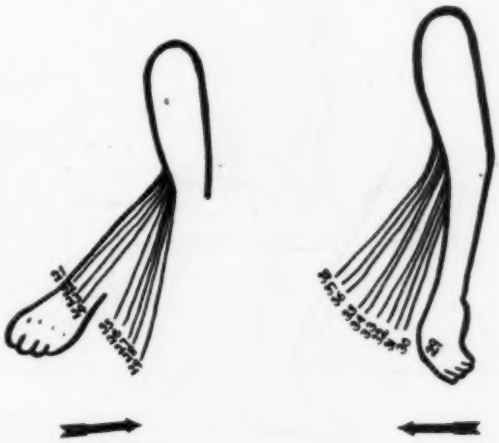

Fig. 11.—This picture illustrates the movements at the elbow as shown by the arrows.

Numerous other similar quotations could be given.

Movements of the Knee.—If a series of tracings of moving pictures is analyzed as far as movements of the knee are concerned, it is found that at the stage when the hip has ceased its backward or ventral movement, the knee is in direct alinement with it (Fig. 13). In the next tracing the hip starts to move forward or dorsally, while the knee starts to move backward or ventrally. This illustrates two points: 1. The ventral position of the leg, as has been previously contended, is one which leaves entirely out of consideration the knee and ankle and controls the leg as a rod. 2. Superimposed on this is the flexion

6. Mackenzie, W. C.: The Action of Muscles, New York, Paul B. Hoeber, 1918.

phase which begins with the process of carrying the leg forward—dorsally. When the ventral movements of the hip have ceased, that is, when the hip starts to move forward, or dorsad, if the movements at the knee are successively traced, it is found that the flexion reflex is at its maximum at a point of time midway between the assumption at the hip of the ventral and dorsal positions, as would be expected (Figs. 9 *B* and 10 *C*). As this point of maximum flexion reflex state is passed, the knee begins to extend until, in the dorsal position, it is again in alinement with the hip, producing a rodlike appearance (Fig. 10 *C*). When the successive stages between the forward (*dorsad*) and the backward (*ventrad*) positions are traced (Figs. 10 *D* and 9 *A*), that is, passing from dorsal through support into ventral, it is found that there is a little flexion at the knee at a point midway between the

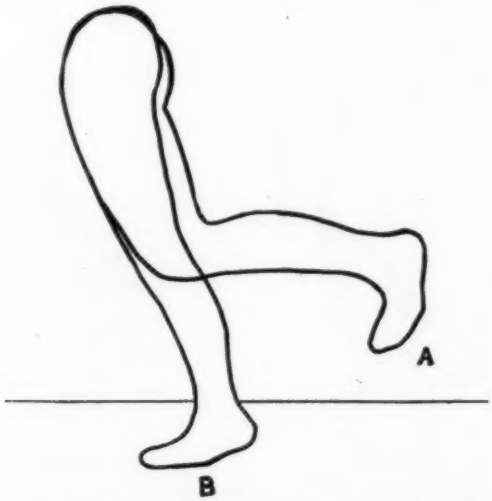

Fig. 12.—This illustrates the change in the location of the posterior thigh line, owing to contraction of the hamstring group of muscles, *A*, and indicates that the anterior thigh line must be used as a constant in comparing successive tracings of the thigh. *A* shows the contour in flexion at the knee; *B*, the contour in extension at the knee.

dorsal and ventral positions and corresponding to the maximum flexion in the other half of the pace. In the sequence ventrad-dorsad it is also found that the hip of the leg of support is directly opposite the hip of the leg of progression (Fig. 5). Thus it appears that:

1. The movement at the hip is diphasic and passes from the ventral to the dorsal position with almost pendulum-like regularity (Figs. 7 and 8).

2. The movements of the knee are tetraphasic. Two knee phases occur during each hip phase (Figs. 9 and 10).

3. The knee phases occurring during the passage of the hip from ventral to dorsal (Figs. 9 *B* and 10 *C*) showed an excursion about twice as great as those occurring during the passage of the hip from dorsal to ventral (Figs. 10 *D* and 9 *A*).

4. On account of its anatomic make-up, though the actual movements carry the foreleg dorsad, the knee never goes into a truly dorsal position.

Flexion of the Knee in the Position of Support.—Furthermore, an unexpected finding has been noted, namely, that the knee is slightly flexed at the phase of maximum support (Figs. 5, 9 *A* and 10 *D*), corresponding to the flexion phase in the other leg and to a phase

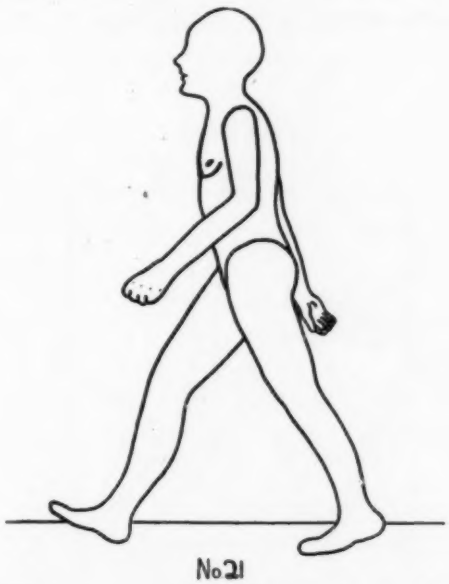

Fig. 13.—This illustrates the ventral position (left leg) and the dorsal position (right leg)—the first two elements of walking. Compare with Figures 5 and 14.

midway between the dorsal and ventral hip positions. If the particular tracing in which this is found is examined, it is noted that there is forward tilting of the torso, that is, that the center of gravity is not the same as in standing but is thrown slightly forward (Figs. 7 and 8). This slight flexion at the knee in the stage of support appears to be an adaptation to the forward placing of the center of gravity. As expected, in running, when the center of the gravity is still further forward, flexion of the knee in the phase of support was observed to be still greater. The oscillations of the knee into this slight stage of flexion (Fig. 10 *D*) and out of it (Fig. 9 *A*) are very much smaller

than those which occur in the flexion phase while passing from the ventral to the dorsal positions (Figs. 9 *B* and 10 *C*).

In the analysis of reflex stepping by Sherrington, previously alluded to, it was stated that in the "extensor" or antigravity phase (support), the knee was extended. In normal standing, this is the case. However, in the process of moving forward the pushing forward of the center of gravity somewhat negates this standing position. The importance of this in analyzing the condition known as decerebrate rigidity or antigravity posture, whether produced experimentally in animals or as a result of disease in man, is considerable; for, whereas the position of the legs in standing represents that reflex which Sherrington calls "the static of erect posture," the positions in walking and running represent the static posture plus a reaction to the forward placing

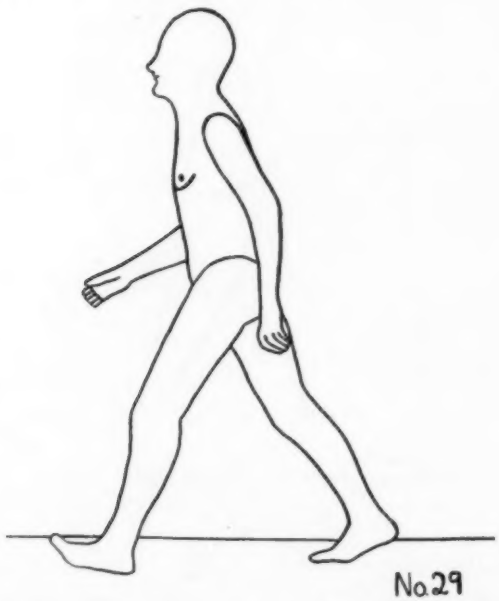

Fig. 14.—This illustrates the ventral position (right leg) and the dorsal position (left leg)—the first two elements of walking. Compare with Figures 5 and 13.

of the center of gravity. The importance in accounting for the flexion of the knees in the posture of parkinsonian syndromes has been alluded to in another article on that subject.[7] The importance of this point in relating postural defects of hemiplegia, pseudobulbar paralysis and the parkinsonian syndromes will be discussed at length another time.

7. Kraus, Walter M.: An Interpretation of the Posture of Parkinsonian Syndromes in Terms of the Neuromuscular Mechanism, New York State J. M. **22**:9, 1922.

However, here it may be remarked that the unilateral defect of the person with hemiplegia represents involvement of the appendicular muscles only. No change of the torso in relation to the center of gravity occurs since the axial muscles having to do with this are not affected. When, however, bilateral involvement occurs, as in the parkinsonian syndrome and pseudobulbar palsy, the axial muscles are affected and the torso bends forward. As a consequence and as a reaction to the forward placing of the center of gravity, flexion at the knee analogous to that seen in the phase of support in walking appears. That group of neurons or integrations of the nervous system which maintains erect posture is apparently affected. The position of the leg in these three conditions is therefore primarily the same. Secondarily, flexion at the knee occurs in bilateral parkinsonian and pseudo-bulbar syndromes, due to the bilateral involvement.

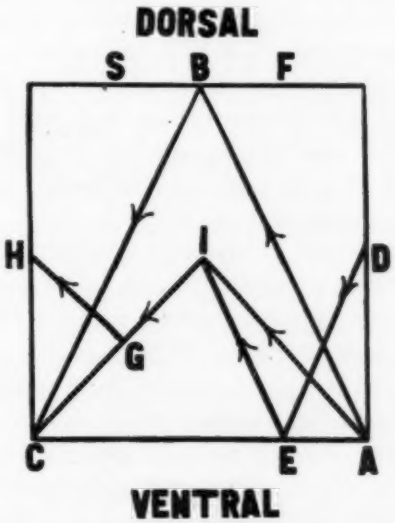

Fig. 15.—Graphic representation of the movements at the hip, shoulder, knee and elbow on one side of the body during a pace of a normal man. *A, B, C,* shoulder and hip. The movement begins at the ventral position, goes to the dorsal position and returns—diphasic; *D, E, I,* flexion phase at the knee—diphasic; *I, G. H;* support phase at the knee—about one-half the extent of *D, E, I*—diphasic. Note that these movements are all from a neutral position midway between ventral and dorsal, into a ventral position and out of a ventral position, back only to a neutral position midway between ventral and dorsal. The knee movement in a complete pace is tetraphasic. *A, I, G, C,* flexion movement at the elbow—diphasic. This shows the same limitations to the ventral field as *D, E, I* and *I, G, H,* the knee movements. The hip and shoulder movements are represented by one line, indicating that though apparently opposite in direction they are really, from a neuromuscular point of view, identical. *F,* maximum flexion; *S,* maximum support.

The position of the arm in the parkinsonian syndrome and in pseudobulbar palsy may easily be shown to be, from the neuromuscular point of view, the same as that of typical contracture in hemiplegia. This may be demonstrated by assuming the position of the arm in the parkinsonian syndrome and then successively contracting all those muscles which have produced this position, namely, the ventral group.

From this it may be seen that the entire hemiplegic position, from the point of view of posture or attitude, represents half of the position of the parkinsonian syndrome and pseudobulbar palsy, minus the reaction at the knee, which, in these latter diseases, is due to the forward placing of the center of gravity.

Identity of Homolateral Movements of the Arm and Leg.—In the earlier parts of this paper it was indicated that by means of the present method of interpretation of movements by the neuromuscular mechanism, it can be shown that the homolateral movement of the arm and leg represents the activity of the corresponding groups of muscles. Reference to the table will show that the leg is carried backward at the hip by a ventral group of muscles. Duchenne [8] has shown that the gluteus maximus does not participate in either walking on the level or standing. This old observation, which I have been able to confirm, forms a final proof that the backward movement or extension at the hip in walking, is entirely ventral.

The forward movement of the arm is carried out by the pectoral muscles, also a ventral group. Thus it is seen that when the leg is carried backward and the arm carried forward both movements are brought about by ventral muscles. When, on the other hand, the leg is carried forward and the arm backward, both movements are carried out by dorsal muscles (Figs. 13 and 14).

At the elbow there is a ventral and a dorsal swing which coincides with the ventral and dorsal swing at the shoulder. The movements are diphasic (Fig. 11) unlike those of the knee, which are tetraphasic (Figs. 9 and 10). The shoulder movements are diphasic like those at the hip.

Figure 15 represents the most important observations of this article in graphic form.

V. SUMMARY

Analysis of the phases of normal human walking, in the light of knowledge of the embryology of muscles, their grouping and nerve supply, has shown that the customary interpretation based on ordinary observation of muscle actions is, in many respects, incomplete and incorrect.

8. Duchenne, G. B.: Physiologie des Mouvements, Paris, 1867.

The homolateral arm and leg, though apparently moving in opposite directions, are moved by the same group of muscles, ventral or dorsal. From the point of view of control by the nervous system, these apparently opposite movements are identical.

The phases of gait are four: (1) the ventral finlike movement of the lower limbs at the hip, (2) the dorsal finlike movement of the lower limbs at the hip, (3) the flexion reflex element, and (4) the support element.

The sequence of these is rhythmic. The order of sequence is 1, 3, 2, 4—1, 3, 2, 4, etc. The first two (1 and 2) represent activity of singly hinged appendages. The second two (3 and 4) represent activity of three-hinged appendages. The first two and the second two each constitute a pair of corresponding opposites. The two elements of the second pair correspond to the flexor (3) and the extensor phases (4) of reflex stepping as described by Sherrington.

In the first two the leg acts like a rod, leaving out of consideration the two large joints at the knee and ankle. The muscular activity of the ventral phase is obviously antagonistic and opposite to that of the dorsal. The alternating muscular activity at the three great joints of the flexion reflex phase is also opposite to that of the support phase.

Analysis of the hip movements shows them to be diphasic. The shoulder and elbow show the same diphasic activity as the hip. Analysis of the knee movements shows them to be tetraphasic. Two phases occur during each hip phase. The two hip phases are from the ventral to the dorsal position and from the dorsal to the ventral position. The two knee phases constituting the flexion reflex element of a pace occur in the ventral to dorsal hip phase. The two knee phases constituting the support element of a pace occur in the dorsal to ventral hip phase.

In the flexion reflex phase the knee passes through an arc about twice as great as in the support phase. At the maximum support stage the knee is slightly flexed. This is due to the forward placing of the center of gravity of the body.

In the typical postural defect of hemiplegia only the appendicular muscles on one side are involved. In the typical bilateral postural defect of parkinsonian syndromes and pseudobulbar palsy the appendicular muscles are involved on both sides, as well as the axial muscles on both sides. The facts given in regard to the forward placing of the center of gravity show that the typical postural defect of hemiplegia represents half of the typical bilateral postural defect of the parkinsonian syndrome and of pseudobulbar palsy, minus the reflex to the forward placing of the center of gravity.

THE NATURE OF CERTAIN FUNCTIONAL NERVOUS DISTURBANCES AND THEIR TREATMENT ALONG METABOLIC LINES*

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It is my purpose to attempt to outline some of the pathologic conditions concerned in certain functional and possibly a few organic disturbances within the field of neurology; and to direct attention to some forms of treatment based on these.

In the course of observation on about 1,000 cases of arthritis, a great many associated clinical disturbances were found. Among the chief of these, so far as the neurologic field is concerned, are fatigue, mental lethargy, headache and migraine, vertigo, tinnitus, neuritis, practically the whole complex of neurasthenia in its many phases and mental depression. In the process of treatment of the arthritic patients, many of the associated neuropsychiatric symptoms disappeared wholly or in part. It is obvious, therefore, that the same underlying causes exist in both groups to a large degree.

To focal infection as the cause of disease has been ascribed too generic a rôle, but there is no doubt that a large number of functional neuropsychiatric disturbances have been shown definitely to arise from such premises.

In considering so-called focal infection and its consequences, however, it is important to realize that a considerable variety of agents or conditions may act in an analogous manner and produce the same results. This broad truth is inadequately appreciated. It was evident, for example, during the studies on arthritis, that in addition to the accepted focal infections within the mouth, head, genito-urinary tract and elsewhere, many diseases, especially dysentery, acted comparably. Exposure to cold and wet was productive of the same consequences and, indeed, constituted the largest immediately precipitating factor. It is important, therefore, to appreciate that the disease processes arise from any interruption in a fairly long chain of established physiologic events, and that even mechanical disturbances of certain broad anatomic relations may contribute to this end.

This chain involves various mechanisms, including the large field of the respiratory functions of the blood, now being subjected to so much study; the ductless glands, particularly the thyroid; the metabolism of

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*Read by invitation before the Philadelphia Neurological Society, Nov. 24, 1922.

the muscular system; the gastro-intestinal tract; and a large number of accessory physiologic acts which normally preserve the balance of health, such as exercise, pulmonary ventilation, perspiration, and the like. There is no opportunity here to discuss at length the dynamic pathologic condition caused by interruption of this chain; but it may be said that there is reason to regard it as arising from interference with oxidative metabolism, using that term in a wide sense.

Some of the reasons for this view are to be seen in the results of the studies on arthritis and focal infection referred to in the foregoing.[1] Omitting negative data which helped to delimit the problem these results were, in brief: a slight lowering of the basal metabolism in 20 per cent. of the cases studied; a high value for blood creatin in about one half of the cases studied, which can be interpreted as evidence of disturbance in the metabolism of carbohydrate; a slight lag in the elimination of water and salt, and a lowering of the sugar tolerance more or less proportionate to the severity of the arthritis.

This lowered tolerance tends to return to normal with convalescence or recovery, but does so most abruptly after the removal of apparently causative focal infection. The contrast under these circumstances may be striking.

It is important to realize that a lowered sugar tolerance is not specific for any disease, even diabetes, and is to be regarded somewhat in the light of a leukocytosis or fever chart. Inasmuch as a lowered sugar tolerance seems to be a function of inflammatory processes at large, it accompanies a considerable range of conditions, and has been made the basis of some too sweeping conclusions. If these limitations and absence of specificity be borne in mind, however, the test becomes valuable for reflecting changes in the so-called dynamic pathology which morphologic methods of study do not reveal.

Starting from this premise, B. M. Hendrix, C. Y. Crouter and I, in collaboration with R. B. Osgood,[2] observed in a long series of normal, arthritic and variously diseased subjects under certain conditions of study, that parallel with the lowered sugar tolerance which accompanies severe expressions of focal infection, there was generally a rise in the percentage saturation of the blood with oxygen. This rise was not reflected in normal subjects, whose sugar tolerance was normal. Furthermore, arthritic patients as a group showed a slightly higher percentage saturation of their venous blood with oxygen than did normal persons or certain other types of diseased subjects; and in some cases

1. Pemberton, Ralph; Robertson, J. W.; Tompkins, Edna H.; Foster, Goodwin L., and Buckman, Thomas E.: Studies on Arthritis in Army Based on Four Hundred Cases, Arch. Int. Med. **25**:231, 241, 243, 335 and 351, 1920.

2. In process of publication in the delayed September number of the Journal of Metabolic Research.

the figures were very high. This high saturation may remain roughly constant over long periods of time. In the light of these data, it is reasonable to deduce that something interferes with the removal of the oxygen from the venous blood as it passes through the tissues. On a number of counts, such as study of the dissociation curves of the blood for oxygen and carbon dioxid, the mechanism does not seem to be chemical in nature, and the thought presents itself that the explanation lies in the rate at which the blood passes through the tissues owing to some change in the peripheral vascular mechanism. This has not been proved as yet, though arthritic patients seem to have a lower rate of blood flow in the hands than do normal patients, as measured by Stewart's method of calorimetry. The real modus operandi, therefore, must await further investigation. It should be stated, as Meakins and Barcroft have pointed out, that certain external conditions, such as great heat locally, may somewhat similarly modify the blood gases in the same direction; but these are not adequate to explain the foregoing findings. Coupled with a slightly lowered basal metabolism and the lowered glucose tolerance, these facts, together with a considerable number of clinical data, strongly suggest that the underlying difficulty relates to some part of the oxidative equation; by this is meant the removal of the products of oxidation as well as the delivery of oxygen to the tissues.

The exhaustive studies conducted on aviators in this country and Europe during the war have demonstrated that some of the symptoms above described, such as headache, mental lethargy, fatigue and variously confused mental states, can be brought about by exposure to conditions of low atmospheric pressure. These experiments are conducted under conditions which simulate, so far as the gases of the blood are concerned, those encountered at high altitudes, the chief factor in which is the anoxemia produced by the lessened amount of oxygen available.

It is interesting to note as a corollary to these findings that the agents which are of most use in combating the effect of focal infection, apart from operative removal of it, are those which in one way or another stimulate metabolism. Indeed, it is frequently possible to observe benefit from some of them to the neurologic complications which accompany arthritis or which may arise, independently of it, from the same causes. This has been particularly noteworthy in relation to headache, mental hebetude and neuritis. The agents in general which are helpful are hydrotherapy, exercise, massage, arsenic, the iodids, radium, thyroid extract, nonspecific protein and some others. In one way or another, these depend for their effect on closely comparable actions, many of which can be demonstrated in the precise terms of the laboratory. The increased metabolism of arsenic and radium can be observed in the urine; and the influence of exercise,

hydrotherapy and thyroid extract is easily demonstrable through calorimetry. Much of the relation of the endocrins to the syndrome under discussion is speculative, but so far as the thyroid is concerned, it is in some part at least exact; and Kendall has adduced evidence to show that thyroxin acts by virtue of removal of the carbon dioxid picked up by one of its benzene rings.

In view of the wide use of hydrotherapy made by alienists in the form of continuous immersion in the hot bath, it is of interest to note the results of some studies by C. Y. Crouter and myself [3] on the response of the body to external heat in the form of electric "bakes." It was found that during the bake the sweat almost invariably changes its hydrogen-ion concentration or "reaction," whatever this may have been at the start. It is generally about neutral at the outset, but becomes more alkaline or less acid as the bake progresses. The most striking exceptions to this have been among arthritic patients whose sweat was more acid than that of any normal person and sometimes showed no changes of reaction, remaining at the same acid point throughout.

It may be added further that if an arm is surrounded by a rubber bag during a "bake," determinations of the carbon dioxid of the air in the bag show that this increases coincidentally with the change in the reaction of the sweat. Whether this is due solely to the increased metabolism of the sweat glands or to an increased elimination of carbon dioxid independently of this, cannot now be stated, but the end-result is an added loss from the body of the chief end-product of combustion. The question is under further investigation. There are other changes of metabolism of much interest bearing on the subject of the therapeutic application of external heat, whose technical nature does not allow of inclusion here.

We thus have two sides to the picture: On the one hand, there is evidence of impaired oxidative metabolism, and on the other, evidence of benefit from the agents which improve this mechanism.

There is still another way by which the body metabolism may be importantly modified. Metabolism is divided roughly into two kinds: endogenous, or that which goes on irrespective of material introduced into the economy; and exogenous, or the metabolism of that which is introduced from without, such as food, with its subsequent reciprocal influence on the body tissues. The therapeutic measures and agents mentioned influence chiefly the endogenous metabolism, although perhaps all phases are affected. The exogenous metabolism is, by definition, open to great modification by changes in the dietary, and there is in this another means of influencing the oxidative equation above outlined.

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3. Pemberton, Ralph, and Crouter, Caroline Y.: The Response to the Therapeutic Application of External Heat, J. A. M. A., to be published.

As previously mentioned, development of the value of the dietary to the syndrome under discussion originated in relation to arthritis, but enlarging clinical experience has shown its applicability to wider fields. There are several ways in which it operates. If the syndrome under consideration is characterized by difficulty in completing the oxidative processes, broadly speaking, it would seem logical that attempts to modify the burden imposed on this mechanism might have a useful influence; and, indeed, experience has shown this to be the case. In general, the benefits from diet under these circumstances are obtained through lightening this burden, that is to say, chiefly through reducing the caloric total of the food ingested. There are differences in the way in which the three foodstuffs act, but whatever the proportions of the foodstuffs, within reasonable limits, the result will be much the same if the total number of calories is reduced. Of the three foodstuffs, protein, carbohydrate and fat, reduction in the carbohydrate is followed by the largest benefits, probably because this substance yields under average conditions, the largest quota of calories, and hence is most concerned in the combustive processes. It is, perhaps, not generally appreciated by those working in fields unrelated to metabolism that nearly 60 per cent. of the protein food eaten becomes carbohydrate within the economy and is combusted in this form. It can be seen from this that often there may be advantage also in reducing the protein intake, though not for the reason popularly accepted that "red meats" in themselves are injurious. There is little or no evidence to indicate that the nitrogenous fraction of the protein molecule contributes importantly or specifically to the pathologic condition mentioned in a strict metabolic sense.

The rôle of the fats is less easy to define, but it is probable that they act in about the same way, although their slower rate of combustion sometimes permits of their larger utilization.

TREATMENT

Treatment, by means of dietetics, of the metabolic disturbances arising out of the agents and conditions mentioned, consists, therefore, essentially in catering to a weakened function, and presents the advantages and limitations of such a procedure. When the reserve of the organism is adequate, lightening the load is sometimes followed by brilliantly successful consequences. If function is much impaired, however, these results may be produced slowly; they may be produced only when abetted by measures which specifically improve metabolic function; or they may not be produced at all.

The situation is in some ways comparable to that occurring in cardiac decompensation. It is obvious that an overtaxed heart may respond to rest alone; or it may be beyond benefit from the lowered demands which

rest alone determines, and may then require further and accessory therapy from digitalis and other measures; or, finally, it may not respond at all to even a combination of measures. The analogy to cardiac decompensation is not complete, since rest is probably always harmless; whereas adjustment of the metabolic burden beyond a certain point may entail varying degrees of undernutrition.

It should be emphasized at this point that treatment of a disease-process on the basis of adjusting a burden to a weakened function should rarely be carried out if the cause of that weakened function can be removed; or in the presence of anemia, marked asthenia, undernutrition, and the like. The very definition of such therapy presupposes that the removal of a burden is accompanied by a capacity to react after such removal. This cannot be too strongly emphasized. There is nothing in these measures which in any way contraverts the important principles of rest and forced nutrition, when indicated, and, indeed, the factor of rest assumes even added importance.

It is my custom, in attempting treatment along this line, to determine the average caloric intake for a given person. This usually requires the cooperation of a dietitian in weighing food and estimating caloric values, but from such data there can be obtained a visualization of the burden on the exogenous metabolism. In instances where this is great, because of a large intake of food with a high caloric total, the metabolism can be spared a large burden by reducing this without detriment to nutritional needs. Thus, if the accustomed intake is 3,000 calories, it is obvious that 1,000 calories can be omitted with entire safety under average conditions. If, on the other hand, the accustomed intake is 1,500 or 1,800 calories or less, as is frequently the case in chronic invalidism, further curtailment must be cautiously made with the fullest regard for nutritive necessities. Complete starvation, accompanied by conditions approximating basal metabolism, is sometimes followed by striking results, but I desire to urge caution against the lax use of such measures. The proper selection of the type of cases and the proper conduction of the measures concerned are the sine qua non of success. Space will not permit me to cite illustrative cases which have been detailed at length in previous publications.[4]

There is another mechanism by which curtailment of diet may have gratifying results. It has been clearly shown by many workers, chiefly in surgical fields, that the colon often plays an initial rôle in causing disease by virtue of absorption of various evil products. Irrigation has been depended on in large measure to further elimination of this material; and while this is useful, it is common knowledge that it often

4. Pemberton, Ralph: The Use of Diet in the Treatment of Chronic Arthritis, Am. J. Med. Sc. **161**:517 (April) 1921.

fails. In this connection, it is pertinent to note that the materials which it is desired chiefly to remove by irrigation are precisely those which are in large part ingested by mouth, and that in cutting down on the dietary we also cut down on the substances producing these undesirable end products. The foodstuffs which give rise to them within the colon are partly, but not exclusively, those which are susceptible to putrefaction, namely, highly nitrogenous foods; and in cases presenting this difficulty, there may be real advantages in withholding such substances as meat and eggs. Carbohydrate foodstuffs may behave similarly, however. On broad grounds it can be easily seen that efforts to meet such a pathologic condition by flushing out the colon is something of an Augean task unless the supply is controlled at the source, a fact which is widely overlooked.

It is not to be supposed that the measures last mentioned, or, indeed, any measures directed toward the neurasthenic syndrome, have universal applicability or that they act as panaceas. If the general nature of the pathologic disturbances under consideration be borne in mind, however, it may be appreciated that the various measures discussed have their respective, though limited, applications, and that results may often be achieved by combining them, where dependence on one measure alone would fail. It is for this reason that the results of correcting or removing "focal infection" are so often disappointing. It is to be appreciated that following long-standing mal-function of various chronic diseases and long-standing focal infection, there may be a more or less permanent dislocation of many normal physiologic processes, which may require time and many accessory measures for restoration. The orthopedists have brought to bear a helpful point of view, in certain cases handicapped by faulty posture or "bodily habit"; but in severe cases restoration by any means may be impossible.

ILLUSTRATIVE CASES

Illustrations of immediate and delayed return to normal of the disturbed physiology described can be seen, for example, in such instances as the following: A soldier of 19 suffering from an arthritis of nine months' duration presented badly infected tonsils and a greatly lowered sugar tolerance. Two weeks after removal of his tonsils his clinical symptoms had entirely cleared up, and the sugar tolerance had returned to normal. Another example was that of a young soldier suffering from an arthritis of the hands and feet. About two weeks after removal of his tonsils, which were badly infected, his clinical symptoms had cleared up, but the lowered sugar tolerance had not fully returned to normal, although it was greatly improved.

A man of 40, presenting spondylitis and abscessed teeth, showed improvement in his general condition and improvement in his lowered

sugar tolerance following attention to these foci, but the sugar tolerance remained at an abnormal level for many weeks.

Finally, several cases which had been watched critically during complete convalescence along the line of restricted diet showed a slow though extensive return of the sugar tolerance to normal. In some patients who recovered after this treatment, the lowered tolerance persisted at its previous level. It is fair to deduce in the latter cases that improvement by this form of treatment was achieved by catering to a lowered function and placing lessened demands on it. The function remained disturbed, but was able successfully to perform smaller amounts of work. Disturbances in the blood gases described can be shown sometimes to follow the same general course.

So fundamental are the changes in metabolism brought about by the various measures discussed that I am prepared for wider application of them than has yet been made. The more fundamental the physiology concerned, the wider the consequences of its disturbance and correction. The processes of oxidative metabolism are essentially the same in whatever tissue they occur, whether nervous, muscular or glandular, and are dependent on the same underlying factors, though their expressions in terms of disturbed function vary greatly.

METABOLISM IN THE NERVOUS SYSTEM

It is of the highest importance to appreciate that the various tissues of the nervous system have an active metabolism in which the processes of utilization of oxygen and production of carbon dioxid and heat are real and measurable. According to Mathews,[5] the need for oxygen is greater in the nervous tissues than in any other tissue of the body. Study of the nervous system in its more precise aspects has been confined almost exclusively to histopathologic and anatomic observations. Students of metabolism, in general, have focused their attention on other and more accessible structures, but it is none the less true that the nervous system, like every other tissue, is dependent on processes of true combustion accompanied by the synthesis and disintegration of tissue. The thoroughness and facility with which these processes are carried out condition the efficiency of the cell unit. A host of factors influences these processes, and the cells in nervous tissue may be laboring under a heavy burden although giving little or no evidences of that to histologic study. These disturbances may arise quite apart from trauma or infection, although it is doubtless true that infection of various kinds operates the more easily on such prepared soil. The work of Child[6] in relation to reestablishment of the youthful type of met-

5. Mathews: Physiological Chemistry, Ed. 4.

6. Child: Am. J. Physiol. **48**:256.

abolism in planaria following starvation, is full of suggestion and, in conjunction with other work along similar lines, corroborates the point of view outlined above.

In few instances are disease processes confined to isolated metabolic errors, as was formerly more widely believed. Normal physiology provides for a considerable margin of substitution and addition in its processes, so that one system can often function somewhat in lieu of another or accessory to it. Though the chain of normal events is long, interruption at one point may be entirely compensated for; but, if not, correction must in general depend on overuse or development of other parts with proportionate adjustment of burdens elsewhere. This is the lesson being taught by the study of diabetes, rickets and the interesting fields of the acidbase equilibrium and the respiratory functions of the blood.

The more precise laboratory methods which sometimes reveal the pathologic disturbance under discussion may occasionally be used for detecting this disturbance when the superficial clinical manifestations of it are slight. Thus, a lowered sugar tolerance or a disturbance in the blood gases, as above outlined, a high blood creatin, and possibly changes in the reaction of the sweat, may sometimes be of diagnostic value in doubtful cases, and may also be used at times as criteria of the return of this dislocated function to normal. It would be improper to place undue emphasis on the information to be derived from this source, but under certain circumstances it may be very real.

SUMMARY

Many forms of neurasthenia, headache, migraine, most cases of neuritis, certain types of mental depression and melancholia—possibly a few supposedly organic conditions—are the result of interruptions in a chain of metabolic processes.

Part of the metabolic process apparently concerned is, broadly speaking, oxidative in nature. By this is meant the delivery of oxygen, the removal of the products of combustion or both. Interruption of the normal course of events may be due to a large number of factors, among which are external physical conditions, the endocrine system, focal infection, mal function of various viscera and various metabolic insults. Appreciation of the general nature of this disturbance makes possible the stimulation of metabolic functions by a variety of measures on the one hand, and adjustment of the metabolic load on the other. A combination of both measures is sometimes helpful or necessary.

Among the few methods of adjusting the metabolic load is that afforded by diet. When applied to properly selected and appropriate cases important results may follow its use. The limitations and indi-

cations in employing it must be critically borne in mind, and at least a working knowledge of nutritional problems is necessary to safety and the best results.

Some of the laboratory findings, such as a lowered sugar tolerance, changes in the blood gas equilibrium, increased values for blood creatin and possibly changes in the reaction of the sweat, accompanying the syndrome under discussion, may, in the absence of a clear clinical picture, serve at times as indexes of the pathology concerned.

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Abstracts from Current Literature

THE SYNDROME OF THE CENTRAL GRAY NUCLEI. George Bickel, Rev. méd. de la Suisse Rom. No. 6: 365 (June) 1922; No. 7: 434 (July) 1922.

Of the gray masses of the cerebrospinal axis, there are none about which we know so little as the central gray nuclei. Only during the last fifteen years has any advance been made in the study of the function and disturbances of these basal ganglions. This advance has been brought about especially by the work of von Monakow and Déjerine. Before beginning this study the author reviews the anatomy of the basal nuclei, this being of importance for the interpretation of the phenomena consecutive to their alteration.

Anatomy of the Central Nuclei.—The author divides the central nuclei into three groups: (1) thalamic nuclei, (2) caudate, (3) lenticular. The two latter are commonly known as the corpus striatum. A study of the optic thalami shows that they are in reality formed by several nuclei, more or less completely fused at certain points. The subdivision of Burdach is the one usually employed—the anterior, the external and the internal nucleus. The external nucleus is called the pulvinar, and by some is considered independent of the optic thalamus. The external geniculate is also considered separate from the pulvinar. Between the external and internal nuclei is a fourth (Luys' nucleus), and finally a fifth, below and in back of the fourth, called the semilunar nucleus of Flechsig. Von Monakow has developed a more minute classification, each of the above nuclei having separate subdivisions, and each division having a cerebral representative. Histologically, the optic thalami are made up of two types of nerve cells; the one with long axis cylinders and the other with short axis cylinders.

The corpus striatum is composed of two separate nuclei, the caudate and the lenticular. The lenticular nucleus is divided into two parts by bands of white fiber. The external portion is called the putamen; the two internal portions, the globus pallidus. The researches of Bielschowsky have shown that the globus pallidus is composed of cells of type 1 of Golgi. The putamen is made up of two types of cells, some type 1, but most small cells of type 2 of Golgi. The caudate nucleus has also two types of cells similar to those of the putamen, with which it fuses in its inferior portion.

In place of the division of the corpus striatum into lenticular and caudate nuclei, recent work tends to the classification of the centers on a histologic basis. The caudate nucleus and external portion of the lenticular nucleus or putamen are identical and are now called the striatum or neostriatum, while the globus pallidus is called pallidum or paleostriatum.

Taking up the connection of the various ganglions between themselves and between the remainder of the brain, the author states that:

I. Union of Central Gray Nuclei Between Themselves: 1. The striatum is connected to the pallidum by the striopallidal fibers. Experimental destruction of the putamen in monkeys, as done by Wilson, shows a degeneration of these fibers, the degeneration going no farther than the pallidum. 2. The thalamus furnishes the only afferent fibers to the striatum. Destruction of the thalamus in monkeys causes a secondary degeneration of these fibers. 3. The pallidum unites with the optic thalamus by a system of afferent fibers, which after traversing the external capsule run to the external side of the thalamus.

II. Union of Central Nuclei with Cortex: 1. The thalamus connects with the cortex through the corona radiata, which also has the corticothalamic fibers coming from all portions of the cortex. 2. It appears established that there is no true connection between the corpora striata and the cerebral cortex.

III. Union of Central Nuclei with Subthalamic Centers: 1. The thalamus connects with the subthalamic regions by three groups of fibers: (*a*) with the nuclei of Goll and Burdach through the tract of Reil, (*b*) with the opposite olives through the superior cerebral peduncle, (c) with the red nucleus and the reticular formation of the cerebral peduncles by the fasciculi of Forel and the radiation of Calot.

There are also connections between the optic thalami and the anterior corpora quadrigemina and the visual apparatus by fibers between the pulvinar and the external geniculate body. There are analogous communications between the nuclei amygdalae and the area olfactorius by the taenia semicircularis; with the mamillary bodies by the fasciculus of Vicq d' Azyr; with the ganglion of the habenula by the olfactory radiations and the fasciculi septothalamici. In most of these connections there are fibers which place the optic thalamus in relation to the motor system by the way of the pyramidal tracts. The existence of these fibers is still doubted.

2. Fibers descend from the corpus striatum in four groups: (*a*) fasciculus pallidorubral, ending in the red nucleus; (*b*) fasciculus pallido-Luysii, ending in the hypothalamus; (*c*) fasciculus pallidonigri, going to the locus niger; (*d*) striotegmental fasciculus, to the nucleus of the posterior commissure and to the nucleus of Darkschewitsch.

The optic thalamus, thus, has numerous connections with the cortex. The corpus striatum, on the contrary, is an organ strictly autonomous, independent of the cerebral cortex, its afferent fibers coming directly from the optic thalamus and the efferent fibers going either to the thalamus or to the subthalamic region.

According to Malone, the pallidum is made up of the motor type of cells; the thalamus, of the sensory type.

Clinical Anatomic Syndromes.—The thalamic syndrome is the best established syndrome of the basal nuclei. Associated with this are the striate and pallidum syndromes.

I. The Thalamic Syndrome: This syndrome was first noted by Déjerine and Egger in 1903. It can be determined best by the development of a tumor, hemorrhage or thrombosis. The symptomatology is usually unilateral, this being a point of distinction from the other basal nuclei syndromes, which tend to be bilateral. The chief symptom is a dissociated hemianesthesia, mild in the superficial sensations but profound in the deep sensibility. There is always an anesthesia dolorosa. The pain is continuous with paroxysmal exacerbations. It may be paresthetic in type (formications, engorgements, etc.), and rebels against all treatment. Hemiataxia occurs. This never attains the grade of a tabetic ataxia, and often must be searched for. The mechanism of the ataxia is due to a failure of the coordinating centers to measure with exactness the motor impulses necessary for the correct production of a motion. If the lesion is near the pulvinar, one frequently has an homonymous hemianopsia of the opposite side. This may also occur if the lesion is in the radiations of Gratiolet or in the external geniculate body. A mild hemiplegia may be associated, usually transitory, accompanied by choreo-athetoid movement. These symptoms may be due to involvement of the internal capsule or striate body. The thalamic syndrome is then a sensory syndrome, characterized by hemianesthesia, hemiparesthesia and hemiataxia.

II. The Strioglobal Syndrome: Under this heading are included: (1) Progressive lenticular degeneration (Wilson). This syndrome was described by Wilson in 1912, though reference had been made to this condition previously. The affection is usually familial, rarely occurring in two successive generations. It appears, as a rule, between the ages of 10 and 25; it progresses slowly, usually terminating in death in from two to seven years.

The characteristics are motor involvement consisting in involuntary movements and hypertonia. The involuntary movements manifest themselves in a tremor, which is the most constant sign. The tremor at first slight, becomes more marked, is rhythmical, most marked in the arms, but may affect all portions of the body. At rest the tremor may disappear; with voluntary movements it is increased. It is an intention type of tremor, similar to that in multiple sclerosis. Some observers describe choreiform or athetoid movements, which are not part of the picture as described by Wilson. It is difficult to say whether the latter signs are truly a part of the syndrome or due to adjacent phenomena.

Rigidity is the most important symptom. It begins insidiously, becomes more marked, appears first in the lower limb, and may spread to all portions of the body. With the increasing rigidity there are contortions, and the limbs and head are held in bizarre positions. Wilson pointed out certain peculiarities which distinguish this hypertonia from ordinary pyramidal spasticity. In ordinary spastic paralysis there are, as a rule, pyramidal signs, such as increased deep reflexes, the Babinski sign and others, all of which are missing in an uncomplicated Wilson's disease. There is also in Wilson's disease a peculiar rigidity of the head, especially of the face, giving the face an odd expression of stiffness and slowness of motion. In the limbs and body the hypertonia creates stiff attitudes, sometimes not unlike tetany. During rest the hypertonia disappears. Power is well preserved, but movements cannot be properly produced.

Usually the rigidity involves also the muscles of phonation and deglutition. The speech becomes altered, it is dragging, scanning and difficult. The muscles of phonation are movable; no trace of atrophy is found; but the patient is unable to talk because of the spasms which hinder him. At times the spasms cease entirely, and the patient may utter a few words intelligibly. Deglutition may be markedly impaired so that even liquids are swallowed with difficulty. Saliva accumulates in the mouth, and there is considerable drooling.

This trio of symptoms — difficulty in speech, deglutition and salivation — suggests a pseudobulbar paralysis. There is, however, no real paralysis, but only the spastic condition. These patients are also emotional; they cry and laugh easily. The gait is disturbed as are all movements. Late in the disease walking may be impossible.

Psychic disturbances are usually present. The patient is dull and slow, at times euphoric, much in contrast to the mental enfeeblement. As described by Wilson, the anatomic lesions are an extreme atrophy of the striatum and pallidum. There is bilateral softening of the lenticular nucleus, especially of the putamen, which may be cystic, leaving the globus pallidus unaffected. There is a destruction of nerve tissue with replacement of neuroglia; later the neuroglia degenerates and cysts are found. Outside of the corpus striatum the lesions are of little importance. The only degenerated nerve fibers are the striofugal fibers which go to the thalamus and the subthalamic nuclei. Wilson insists that the lesions are entirely in the extrapyramidal motor system, tracts going from the striate nuclei to the red nucleus and continuing into the

spinal cord by the fascicule rubrospinal of Monakow. The sensory tracts and pyramidal system are always intact. A hobnailed liver is always associated with the cerebral pathology. The liver picture is especially interesting as it never causes the clinical picture of a cirrhosis. The spleen is hypertrophied. All other organs are normal.

This syndrome, according to the author, merits a special place in pathology and, as Wilson stated, is a new disease.

(2) The Pseudosclerosis of Westphal-Strümpell: This affection closely resembles Wilson's disease. It is characterized by gross trembling, speech disturbance and marked spasticity. It was first described by Westphal in 1883. It was not until 1912, however, that Hoesslin and Alzheimer described the lesions of the central nervous system. In summary, one has an affection characterized by a course tremor of the entire body, exaggerated by voluntary movements and by emotions. There is a muscular rigidity, less marked than in Wilson's disease, a gait and speech disturbance and mental changes which may progress to dementia. As in lenticular degeneration, there is a change in the liver and spleen. Finally, in all cases one finds a ring of brownish-green pigment at the periphery of the cornea, which does not exist, as a rule, in Wilson's disease. In the nervous system, one finds a glia proliferation with partial atrophy of the nerve structures, especially the ganglion cells. These alterations are especially noted in the basal ganglions, as the corpus striatum and hypothalamic region. Changes occur in the cortex, cerebellum and stem. There has been considerable discussion as to the classification of Wilson's disease and pseudosclerosis. The author discusses the various views but comes to no conclusion. As to the etiology of these conditions, little can be said. At present the conditions may be looked on as a constitutionally congenital anomaly, heredofamilial in type.

(3) The Syndrome Pseudobulbar Senilus: In connection with Wilson's disease, it is well to mention the pseudobulbar paralysis of the old, in which there is sometimes a bilateral sclerosis of the lenticular and caudate nuclei. The type of lesions and their location naturally provoke identical symptoms.

III. *The Partial Striate Syndromes.*—In certain conditions lesions are confined to either the caudate or lenticular nucleus, or to the caudate nucleus and putamen or pallidum. Their syndromes are classed as follows:

A. Striate Syndromes (Choreo-Athetoid Syndromes).

(1) Double Athetosis: This was first described by Anton in 1896. The credit for a clear description of the disease belongs to C. Vogt. The affection begins in infancy, is progressive, but usually becomes stationary about the eighth year; it may even become regressive. Clinically there is a bilateral hypertonia associated with athetoid movements, sometimes choreo-athetoid. The speech is of a pseudobulbar type, with spasmodic crying and laughing. In appearance it resembles Wilson's disease, but is distinguished by the athetosis, absence of tremor, its early onset, absence of cirrhosis and difference in cerebral lesions. There is an atrophy of the caudate nucleus and putamen (état marbré du striatum).

(2) Chronic Chorea (Huntington's): The clinical symptoms are well known, and divide themselves into two large groups—progressive mental deterioration and involuntary motor disorders. Jelgersma, in 1908, attributed the disease to an atrophy of the caudate nucleus, which was confirmed by Alzheimer in 1911. Marie and Lhermitte have done the most important work and established that the pathology is an atrophic degeneration of the cortico-striate fibers. They found an atrophy of the caudate nucleus and putamen,

also an atrophy of the cerebral cortex, especially in the frontal and rolandic regions, and, finally, slight changes disseminated throughout the brain. The motor condition is thought to be due to the lesions of the striatum; the psychic manifestations, to cortical changes.

(3) Acute Chorea (Sydenham's): In acute chorea there is an extensive encephalitis. The chief damage occurs in the caudate nucleus. It is practically impossible to differentiate between the lesions of an acute and a chronic chorea.

(4) Chorea and Athetosis in Hemorrhagic Lesions and Softenings: Hemorrhages and softenings, when situated in the striatum, may cause choreoathetoid movements. Such cases seem to be rare.

B. Syndrome of the Pallidum (Parkinsonian Syndrome).

Lesions of the pallidum have long been considered as the cause of paralysis agitans. It is now more or less accepted that the so-called Parkinson's disease is not a morbid entity, but a syndrome, the result of a variable etiology, and due to pathologic changes in similar portions of the brain. Clinically, the syndrome has three chief components: muscular rigidity, abolitions of automatic and associated movements, and a slow tremor which may involve any or all portions of the body, even the vocal cords. Sweating, salivation, paresthesias and cramps may occur. The psychic remains intact, although a certain exaggeration of emotivity may be present.

According to Souques, five types of parkinsonism are distinguished: (*a*) senile or presenile type (classic form), (*b*) juvenile type, (*c*) paralysis agitans without agitation, (*d*) parkinsonian syndrome due to hemorrhage or softening, (*e*) a postencephalitic Parkinson's syndrome.

While it is accepted that the chief pathologic changes occur in the globus pallidus, there is still some room for discussion; thus Tretiakoff is of the opinion that the chief changes occur in the locus niger. The former is, however, the accepted view.

With the foregoing review, the major syndromes of the lesions of the central nuclei have been described. In passing, the author mentions a few other syndromes but does not elaborate, stating that too little is known about them: disintegration and dysmyelinization of the stria (Vogt), dystonia lenticularis of Spiller, arteriosclerotic muscular rigidity (Förster), pyramidopallidal degeneration syndrome (Lhermitte and Cornil), spasmodic pseudosclerosis (Jacob).

Pathologic Physiology.—While the clinical anatomic experiences have tended in a measure to clear away some of the mystery surrounding the central gray nuclei, experimental physiology has tended to complicate the picture. Unfortunately, all experimental results have been so variable and the problem so difficult that the results thus far have been unsatisfactory.

General Conclusions.—In conclusion, the author states that the symptoms of the thalamus are essentially sensory, the manifestations of the corpus striatum purely motor. The rôle of the optic thalami is that of a relay station in the sensory system. They make a definite break in the sensory pathway. Sensations coming from the periphery are, so to say, sorted out here. Some continue their way to the cerebral cortex, where they cause voluntary reactions—reflex movements of special exactness. The others pass to the corpus striatum and produce reactions essentially automatic or elementary. The corpus striatum may be considered as an automatic center, entirely independent of the cortex. Its alterations produce a disorder of muscular function, which is characteristic of the various striate syndromes. All the fundamental ele-

ments, muscular rigidity, attitude, loss of associated movements, marked physiognomy, spasmodic movements and athetochoreic movements, are due to disturbance in the coordinating automatic motor system.

The author feels that the foregoing is a fair summary of our present knowledge of the central gray nuclei. A fruitful harvest may be the result of further study in this fertile field.

MOERSCH, Rochester, Minn.

STUDIES IN FOCAL INFECTION: ITS PRESENCE AND ELIMINATION IN THE FUNCTIONAL PSYCHOSES. NICHOLAS KOPELOFF and CLARENCE O. CHENEY, Am. J. Psychiat. **11**:139 (Oct.) 1922.

THE ETIOLOGY AND TREATMENT OF THE SO-CALLED FUNCTIONAL PSYCHOSES. SUMMARY OF RESULTS BASED UPON THE EXPERIENCE OF FOUR YEARS. HENRY A. COTTON, Am. J. Psychiat. **11**:157 (Oct.) 1922.

One could scarcely find a more strikingly effective instance of the "deadly parallel" than is presented by the simultaneous appearance of these two contributions to the theory of focal infections as etiologic agents in the production of "functional" mental disease. It is fitting and logical that the two articles be reviewed together, yet the task is neither a simple nor an enviable one. No amount of careful and unbiased analysis makes it possible to evade the inevitable conclusion that attacking an identical problem with practically the same weapons, the authors have nevertheless arrived at diametrically opposed conclusions. It would seem proper first to summarize the actual work accomplished by each observer, and then to add the comment which seems to be suggested and justified by the investigations themselves and the remarks of those who took part in the discussion of this important topic.

Kopeloff and Cheney sought to solve the problem of the existence or nonexistence of a causal relationship between focal infections and psychoses by carrying out a carefully controlled therapeutic experiment. In the control group, totaling thirty-three, there were fifteen instances of dementia praecox, fifteen of manic-depressive disease and three psychoneurotic patients; while the group of twenty-seven in whom sources of infection were attacked by surgical procedures was made up of seventeen schizophrenic, nine manic-depressive and one psychoneurotic patient. Care was exercised in selecting the two sets of patients so that they were well-balanced in respect to age, sex, duration of psychosis, diagnosis, prognosis and infective condition of teeth and tonsils. For instance, from the standpoint of dental pathology, 101 teeth in the "controls" were pronounced septic, while in the "operatives" 139 were extracted. All but two of the latter group had had extractions performed and, in addition, sixteen had had tonsillar enucleations. The two questions which the investigators attempted to answer were: "What do these groups of cases show comparatively with respect to recovery, improvement, or unimprovement?" and "Has the removal of focal infections in teeth and tonsils brought about recovery or improvement in individual cases?" The table which is here reproduced would seem to justify the conclusion of the authors that "On the whole, then, the operated group appears to have received no more benefit than the control group." Before attempting to answer the second query, and indeed before actual estimations as to the extent of the focal infections were undertaken, Cheney recorded a diagnostic-prognostic survey from the standpoint of clinical psychiatry. These opinions were not consulted in pro-

nouncing judgment on the end result, but a subsequent comparison showed "that no recovery has taken place that was not prognosticated before any focal treatment was employed. We have no evidence on which to base a conclusion that the removal of focal infection has of itself brought about recovery."

Since he had not undertaken an experimental study, Cotton's article is justifiably more general in its tone. He has previously written in terms specific enough, and his published results in individual cases are known to every student of psychiatry. Since 1918, his recovery rate in the "functional" group (manic-depressive, dementia praecox, paranoid states, psychoneuroses) has averaged 80 per cent. Complete faith in the specific effectiveness of detoxication treatment (by which is implied the surgical treatment of infected teeth, tonsils, uterus, seminal vesicles, colon, etc., plus the injections of gastric autogenous vaccines and antistreptococcic and anticolon serums) is strongly reiterated. Summaries of stomach analysis in 106 psychotic and fifty-six nonpsychotic persons are presented. The following sentence strikes the keynote of the conclusions: "We have produced evidence, both clinical and pathological which should set at rest any doubt as to the accuracy of our deductions."

Granting that Kopeloff is a competent bacteriologist and that Cotton has reliable laboratory facilities, one is faced with a mass of conflicting technical evidence. For instance, Kopeloff found it a matter of great difficulty to secure

Results With and Without Operation

	Dementia Praecox		Manic-Depressive	
	Controls	Operatives	Controls	Operatives
Number of cases	15	17	15	9
Recovered	..	..	5	4
Improved	5	5	8	1
Total benefited	5	5	13	5
Unimproved	10	12	2	4
Left hospital	3	5	6	3

cultures from uncontaminated teeth. No organisms were isolated from the tonsils which are not present in the mouths of healthy persons. The value of the Rehfuss method of gastric analysis as a demonstrator of foci of infection is seriously and pointedly questioned. Furthermore, he believes that the bacterial content of the stomach is influenced more by the swallowing of saliva than by the degree of acidity. The simultaneous aspiration by three Rehfuss tubes gave varying degrees of acidity. It seems that the use of a dental suction pump to take up the saliva reduced the number of stomach bacteria from 48,000 to 32 per cubic centimeter. Cotton not only makes a categorical denial of scientific verity of these objections, but particularly calls attention to contaminating organisms which he himself did not encounter, and he insists that Kopeloff's series of examinations was entirely too small to be worth while. In only a few of Cotton's patients did the organisms in the stomach correspond to those in the mouth. One of the members in his discussion called attention to Schottmüller's inability to find any infective condition which he could ascribe to oral sepsis in 10,000 bacteriologic examinations (*Deutsch. med. Wchnschr.*, Feb. 9, 1922). Doubtless, expert opinions to the contrary could be as readily quoted. While it is possible that the answer to the much mooted question is to be found in the bacteriologic technic, it may not be decided by the opinion of one or two or even several men, and must be eventually answered by evidence still to be accumulated.

What is the present position respecting the rôle of focal infection in psychiatry? How are we to decide whether we are confronted by a theory or an established fact? Kopeloff's and Cheney's well-balanced and carefully controlled experiment should be given some weight. While it is true that they worked with small groups, they were scarcely small enough to make it possible to attribute the negative result to mere chance. Some investigators feel that they have been able to cure their patients; others complain of failure. Nothing is more difficult than the decision as to just what agent or agents contributed to a recovery in a given case. The issue is heavily clouded by the possibility of diagnostic errors. Cheney's preliminary diagnostic-prognostic survey was a wise precaution, which should be repeated in further studies. A benign psychosis may readily be mistaken for a malignant one and vice-versa, but the margin of error may be decreased by an initial strict rating from the standpoint of clinical psychiatry.

Cotton's reported results are brilliant. For four years the recovery rate has averaged 80 per cent. The obstacles in the way of accurate follow-up investigations are always tremendous. In dealing with large groups and with the limited number of field workers available, it is hardly possible to accumulate a large amount of specific information. The type and extent of recovery is often a question of medical judgment, and many patients are able to live outside a hospital even though they are far from normal. Four years is too short a time after which to pass final judgment. Manic-depressive psychosis is notably recurrent, and the clear intervals may be long or short, while schizophrenia is far from being in every instance a continuous psychosis. There are at times well marked remissions and not altogether infrequently a cessation of the psychosis or at least of its outstanding symptoms, with or without appreciable defect. To sum up: A clear-cut negative experiment, the lack of agreement among competent observers, the possibility of errors in the follow-up work, the insufficient length of time which has elapsed since the detoxication treatment was put into effect on a large known group, and the uncertainty of certain aspects of the bacteriologic technic are in favor of the opinion that focal infections are not yet to be regarded as specific etiologic agents in the production of psychotic states; and it seems neither ultraconservative nor unscientific to believe that the problem is still in its embryonic stage.

If, as seems to be the case, the majority of neuropsychiatrists rightly prefer to regard the effect of focal infections as problematical, it seems pertinent to inquire into the usefulness of the theory. What are its advantages? Is it guilty of any sins of commission or omission? While each one must trust his own judgment, there is apparently a fairly large group of cases in which no question can arise. I refer to the patients in whom there is a pathologic condition demonstrable by recognized and more or less standardized methods. It seems reasonable and even necessary to remove such an unquestionably pathologic condition whenever possible. Here psychiatry owes a debt to Cotton and his followers for the emphasis which they have placed on the factor of focal infections. There is no good reason for neglecting to remove teeth and tonsils which are indisputably diseased. On the other hand, enthusiasm should not be permitted to outpace judgment, even when the consideration is the removal of a single tooth. Further, any operation which is attended by a mortality of 30 per cent. should not be undertaken unless the indications are clear-cut and definite. It is not an easy matter to decide that the colon is infected. To argue that death from colectomy is preferable to terminal dementia

is scarcely admissable. Clinical psychiatry has not yet attained that diagnostic perfection which makes it possible to predict terminal dementia with any degree of surety, at least not until the psychosis has reached a stage in which even colectomy will not avail.

A new theory never strengthens itself by a display of impatience with other theories. Even the study of heredity, which is dismissed as a "fixed quantity in the equation," has served psychiatry usefully in the past and has not yet outlived its usefulness. Whether or not it has an influence on the prognosis of a given case is a question which seems honestly debatable, but that it does have a far-reaching effect on the mental health of the race is not to be denied. The ardent advocates of the importance of heredity should at least be credited with having the same objective as do the believers in the theory of focal infection; namely, the reduction of the total percentage of mental disease.

Likewise, the psychogenic factor may not be dismissed with two short pages. One need not hold a brief for the psychogenetic conception to feel that not infrequently a strong emotional stirring, or perhaps even more often the accumulation of many less dramatic stresses, may have been the factor which conditioned the departure from reality. It seems unfortunate to revive the age-long question of structure and function, and one may readily agree with Cotton that the concept of a disordered mind and a perfectly normal brain is untenable. However, one might want to specify that the brain like other organs is capable of ephemeral as well as permanent changes, of chemical disturbances as well as organic pathologic alterations. Instead of stating that "disordered function must depend upon and be the result of disordered structure," one might prefer, for the present at least, to say that "disordered function is associated with disordered structure." Experimental physiologists have produced at least temporary structural alteration by the stimulus of an artificially induced emotional state.

It is scarcely fair to imply that psychiatrists who have not been able to accept all of Cotton's views are standing pat "on a do-nothing-policy." There is much that is worth while in the theory of focal infections, and its useful application is not being neglected. There are also elements of importance in other theories which are being turned to good account. In the last analysis, it will probably be necessary, valuable and constructive for some time to come to regard psychoses as nonspecific reactions. In this way, the problem may be attacked from every possible angle and the door of investigation kept open to its widest possible extent. One might remark that the slogan "focal infections or nothing" will not gain many adherents for the theory.

It is regrettable that the public has been taken so freely into medical confidence. With due respect for the value of public opinion, it is scarcely fitting that it be the arbiter of a question which involves so many technical considerations. "Discoveries" in medicine and perhaps particularly in psychiatry should not be broadcasted in the lay press until every reasonable doubt has been removed.

Many years before Cotton gathered together the available data and emphasized their importance, many neuropsychiatrists were carefully scrutinizing their patients from the organic point of view and correcting correctable physical defects, and they continue to do so. In spite of extremists, there has been a healthy tendency in psychiatry to take advantage of newer methods either developed in our special field or borrowed from the armamentarium of inter-

nal medicine, and there need not be the slightest concern that this broad approach will ever be abandoned. The question of focal infection is being and will be thoroughly tested along with other conceptions, and will eventually be accorded its proper position in neuropsychiatry.

It is scarcely a valid objection to state that many public institutions do not have proper laboratory facilities. Neither do they have facilities for the practice of psychanalyses or the study of endocrine dyscrasias, but the reason is obvious. Surely it is not because they have conceived a strong prejudice against the study of focal infections or psychanalysis or endocrine disorders. Fortunately, there are enough institutions, both public and private, in which intensive investigations are carried out in all fields, and a sufficient number of intelligent neuropsychiatrists who study their patients thoroughly from every point of view, to make it impossible for the theory of "focal infections" as causative factors of psychoses to fall into the discard through lack of scientific attention.

STRECKER, Philadelphia.

OCULOGASTRIC AND OCULOCOLIC REFLEXES. DANIELOPOLU, RADOVICI and CARNIOL, Ann. de méd. **11**:143 (Feb.) 1922.

The authors have shown in previous papers that ocular compression produces, in addition to the oculocardiac reflex, a series of reflexes relating to all the abdominal viscera. They have described an oculo-colic and an oculovesical reflex. The most advantageous cases for the study of these reflexes are those in which there is a destructive spinal lesion, which induces in the sublesional cord a pronounced automatism. With the automatism of voluntary muscles, they have shown that there is a visceral automatism for all the organs dependent on the motor nerves of the cord below the level of the lesion.

The distention of the bladder produced experimentally by insufflation, produces a contraction of the muscles of the abdominal wall (and of the thigh). A similar visceromotor reflex applies to the descending colon.

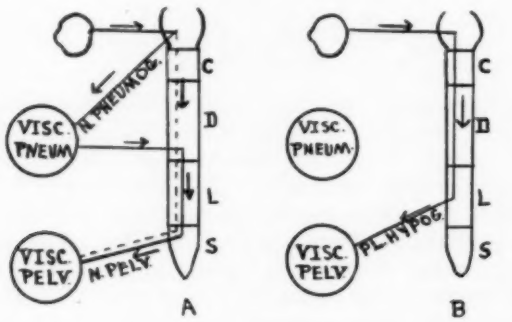
Either pricking or pinching the skin below the level of the lesion gives a cutaneovesical reflex. This excitation of the skin provokes contractions of the bladder wall, accompanied by a desire to urinate. A more intense excitation of the skin, below the lesion, results in both the cutaneovesical reflex and automatic movements of the legs. All excitation of the skin of the trunk superior to the lesion or excitation of the skin of cranial nerve innervation does not cause this reflex.

However, ocular compression, even very slight, brought about an oculovesical reflex in the subject with the spinal lesion; the bladder as well as the descending colon contracted with violence, phenomena which they have been able to record by the graphic method. If the compression is strong or prolonged, after the vesical contraction, automatic movement of the legs occur, movements which the subject is incapable of executing voluntarily.

The authors then undertook to demonstrate whether these reflexes are present in the normal person. They used in their experiments a person whom they choose to term amphotonic, because he showed hyperexcitability of both the vagal and sympathetic systems.

The tracings obtained and published with the paper show that ocular compression produced an oculocolic and oculogastric reflex. This reflex generally displayed itself in two phases, a phase of inhibition followed by a phase of exaggeration. The inhibition related both to tonicity and contractility. Likewise, the phase of exaggeration related to both. But for the stomach, it is

especially inhibition that is produced, and the phase of exaggeration which follows is represented chiefly by increase in tonicity and less by augmentation of contractility. They also found that for the stomach the ocular compression produced an inhibition longer in duration directly in proportion as the contractions prior to the compression had been feeble, but that intense ocular compression made the stomach quiet during a very long phase even if, up to that moment, its contractions had been strong. Thus, it appears that ocular compression provoked an enormous slowing of the contracting rhythm, in direct proportion to the intensity of the compression. In brief, the authors agree that the ocular compression has opposite effects in the different organs; an effect predominately sympathetic (inhibitory) on the stomach and an action predominately vagal (inhibitory) on the heart. They further conclude that the oculovisceral reflexes are conducted by two paths, the sympathetic and parasympathetic (vagal), and that the results obtained depend on the respective



A, possible path of the oculoparasympathetic reflex of the colon; *B*, probable path of the oculosympathetic reflex of the colon.

tonus of the two autogonistic groups of each organ. The reflex is carried by each path to each organ, but predominates on the nerve which presents the higher inherent tonus.

The remainder of the paper deals with a study of the nerve pathways concerned and can be summarized by the diagram presented.

DAVIS, New York.

CLINICO PATHOLOGICAL NOTES ON SOLITARY TUBERCLE OF THE SPINAL CORD. WILLIAM THALHIMER and GEORGE B. HASSIN, Jour. Nerv. & Ment. Dis. **55**:161 (March) 1922.

A man, 28 years old, had been complaining of backache and inability to bend his back for about a year. In the course of eight months he developed intermittent sharp and dull pain in the spine between the shoulder blades, with gradual progressing spastic paralysis of the lower extremities associated with a feeling of numbness. There was a sense of numbness in the rectum and bowels and no knowledge of the flow of urine. The reflexes of the lower extremities were increased, more so on the left; the right abdominal reflex was absent. On the right side of the trunk, there was impaired sensation to pain and touch below the seventh rib; on the left side, a band of hyper-

esthesia at the level of the umbilicus was present. Serologic findings were negative. Roentgen-ray examination revealed osteophytes of the entire lumbar and eleventh and twelfth dorsal vertebrae. The spinal fluid on a few occasions revealed a typical Froin syndrome. A diagnosis of spinal cord tumor was made and enucleated at operation, at the level of the second dorsal vertebra. There was no improvement of the patient's condition, and death followed a few weeks later.

Necropsy Findings: These were: tuberculous scars at the apexes of both lungs, bilateral iliopsoas tuberculous abscess and osteo-arthritis of the spine which the authors regard as probably tuberculous in origin, but a careful examination of all the bodies of the vertebra revealed no tuberculous lesions. The tubercle was made up of numerous miliary tubercles with fibrous formation and cheesy degeneration. All ectodermic tissue was displaced by the tubercle. There was typical secondary degeneration of the spinal cord above and below the tuberculous mass. There was extensive leptomeningitis, pachymeningitis, and peripachymeningitis with involvement of the spinal roots, especially marked at the level of the spinal cord harboring the tubercle. The subdural and subarachnoidal spaces were entirely obliterated. There was total absence of inflammatory phenomena of myelitis in the areas not affected by the tuberculous process.

The authors' conclusions drawn from the pathologic study of this case are: that a solitary tubercle of the spinal cord is the outcome of a local inflammation of the tissues brought on by a specific micro-organism; that the inflammation provokes reactive phenomena on the part of the surrounding tissue elements causing proliferation of the adventitial cells and of hematogenous elements (lymphocytes, plasma cells, etc.); that the adventitial cells, filled with the detritus taken from the inflammatory area, are drained into the subarachnoid space where they provoke various inflammatory reactive phenomena on the part of the pia-arachnoid, that is, a meningitis. It is the authors' claim that the latter is a secondary process, and not due to direct invasion of the meninges by the tubercle in the spinal cord; likewise, that the draining of the disintegrated material by the subarachnoid space as well as the powerful connective tissue rings around the tubercle are responsible for the preservation of the rest of the spinal cord and the absence of myelitis.

The authors have been able to collect sixty-seven reported cases from the literature, which appeared to be definitely proved cases of solitary tubercle of the cord, fifty-eight of which are abstracted in their article. In reviewing these, it has been noted that many of the tubercles were multiple and some situated in the brain and brain-stem as well as the cord. Inflammation of the meninges was associated with it in the majority of cases.

STACK, Philadelphia.

NEO-ARSPHENAMIN IN HYPODERMIC INJECTIONS. MINET and LEGRAND, Rev. de. med. **39**:230 (April) 1922.

The authors recall that the first injections of neo-arsphenamin were made into the muscles. In acid or alkalinized solution, it was injected in the supero-external portion of the buttock, above the great trochanter near the iliac crest. Such injections were excessively painful and produced great lesions of infiltration or necrosis. To obviate this, oily mediums were tried, but because the absorption of such mediums was poor, they did not become a vehicle of choice. When they were abandoned the method of intravenous injection rapidly

increased. The discovery of neo-arsphenamin in 1912, by Ehrlich, simplified the matter as did Ravaut's demonstration that even the largest dose of neo-arsphenamin after being dissolved in the small amount of distilled water which the ampule itself can hold, can with safety be injected intravenously with a small hypodermic syringe and needle.

In spite of this consideration and the increasing inclination to use the veins for medicinal injections, the authors do not find themselves able to ignore the several disadvantages which they feel are inherent in the intravenous method. One disadvantage is the serious trouble that one sometimes has in finding a difficult vein, and the disturbing effects if even a small portion of the solution is allowed to flow outside the vein. Another disadvantage is the accidents, immediate or late, which may result. Mentberger, in 1914, published a statistical report of 229 cases of death from intravenous injections.

More frequent are the so-called nitritoid crises of Milian, a vasomotor syndrome accompanied by a syncopal or lipothymic state. Other accidents assume a gastro-intestinal form—violet gastralgia, sudden diarrhea or vomiting. Sometimes fever, chills, cephalalgia and a so-called Herxheimer reaction develop. Later one observes albuminuria, cutaneous eruptions and late nervous phenomena.

In spite of the widespread use of the intravenous method, therefore, the authors have not rallied to it; instead they early became the protagonists of the method of subcutaneous injection of neo-arsphenamin which Sicard, in 1920, thoroughly reviewed. They believe that by this method they can assuredly avoid nitritoid crises and all the other occasional late and early disagreeable reactions. Further, they are convinced that they can realize a more intensive treatment than is possible with intravenous injections. Sicard never goes beyond 0.15 gm. for an injection. Trossarello does not hesitate to give 0.75 gm. The authors use from 0.05 to 0.30 gm. In one case, desiring a very intense action, they injected 0.45 gm. daily, without any bad effect.

The technic followed is that described by Wechselmann and used also by Sicard. The place chosen for the insertion of the needle is the external surface of the thigh, principally the trochanteric region. The neo-arsphenamin is dissolved in 0.5 to 1c.c. of dissolvent. It is necessary to push the needle so that the injection is made immediately superficial to the fascia lata. To establish this, one needs to find the level at which the needle can be readily displaced in all directions without catching in the trabeculae of the subjacent aponeurosis. Finally, it is necessary to perform the procedure rapidly.

After experimenting with various dissolvents, they found that a glucose solution—47 parts to 1,000—highly satisfactory, but more recently they have used with even better results the glucose solution with phenic acid, the exact proportions of which are not stated. In their paper, the authors also make clear that in conjunction with these injections, they simultaneously subject the patient to mercurial and iodid treatment.

DAVIS, New York.

A TRIAL OF THORIUM X IN PSYCHIATRIC THERAPEUTICS.

P. DADAY, R. BESSIÈRE and L. JALOUSTRE, Presse méd. **30**:520 (June 17) 1922.

This substance loses half its strength about every three and one-half days, and is practically inactive after twenty days. One result of this property is the absence of cumulative effects. The preparation used in the experiments was an isotonic solution of bromid of thorium X, put up in ampules contain-

ing a variable quantity of the active substance, from 10 to 1,000 micrograms in strength. Injections could be given subcutaneously (the method of choice), intramuscularly or intravenously. The drug might also be administered by mouth, if not over 150 micrograms were given in a day.

Each patient was given a series of five injections, one week apart, then after four weeks' rest five more injections were given spaced as before. No dose consisted of less than 350 or more than 700 micrograms.

Local pain was occasionally complained of for several hours after an injection, and was accompanied by a transitory elevation of temperature. Nausea and faintness were sometimes evident. During the second course of injections, almost all the patients exhibited a local or a more or less general pigmentation of the skin, which disappeared in about a fortnight after the last dose. There was also a concurrent tendency to slight anemia and loss of weight. Two patients developed mild ulceration of a finger.

Three groups of patients were treated, diagnosed as having (1) involutional melancholia, (2) dementia praecox, and (3) acute confusion. No improvement was observed in the first group (nine cases). In the second group, the two patients with paranoid praecox and the four of simple praecox of long standing were unimproved, while one with recent simple praecox was apparently cured. The third group comprised three patients, two of which were cured and one improved.

The authors are confident that their confused patients recovered far more rapidly with this treatment than they would have done without it. It was shown that if improvement was to take place in a given case, it would be well marked after the second or third injection. The value of a second series of injections remains doubtful. No theories are offered on the mechanism of the cure.

HUDDLESON, New York.

THE PROCESS OF INGESTION IN THE CILIATE, FRONTONIA.

WILLIAM M. GOLDSMITH, J. Exper. Zool. **36**:333, 1922.

Observations and experiments were made on the ciliate, *Frontonia leucas*, while the organisms were ingesting euglenas, diatoms, desmids and oscillatoria filaments. The ingestion of blue-green algae, especially oscillatoria filaments, furnished the most conclusive demonstrations of the method involved, as the process continued a greater length of time and involved more factors than did the ingestion of smaller organisms. Five factors were involved in the process of ingestion of material longer than the expanded width of the body of *Frontonia*. In case of smaller particles, the third, fourth and fifth factors mentioned below are not essential to ingestion.

(*a*) Action of Oral Cilia: The cilia about the mouth of the frontonia exert a direct pull on the incoming food.

(*b*) Action of Locomotor Cilia: The cilia of the body in general drive the organism forward and thus force the stationary food into the mouth.

(*c*) Rotation of Body Axis: The end of the fiber usually enters the mouth and passes anterodorsally until it comes in contact with and exerts a pressure on the aboral wall, after which the frontonia swings around through an angle of almost 180 degrees, using the mouth as a pivot. This change of position permits the fiber to pass dorsally along the aboral side of the ciliate. Through the play of either factor *a* or *b*, or both, ingestion continues until the fiber exerts such pressure on the body wall at the extreme posterior end that the organism is extremely elongated and pointed. Such pressure on the body wall

acts as a stimulus, causing movements that relieve the stimulation. The rotation of the body axis assists in relieving the stimulation at certain of these tension points.

(*d*) Body Contractions: A series of sharp contractions of the body wall assists in relieving certain other tension points.

(*e*) Cyclosis: Cyclosis aids by moving the end of the fiber around the wall, thus making further ingestion possible.

Unusual and fantastic figures are produced through the contortion of the organism by the ingested food which varies in size, shape, density, elasticity and color.

WYMAN, Boston.

AN EXPERIMENTAL STUDY OF THE MECHANISM OF HALLUCINATIONS. MORTON PRINCE, J. Psychol., Med. Sect. **2**:165, 1922.

Postulating that artificial hallucinations or "crystal visions" are identical with many of the hallucinations of the insane as well as of the sane, Prince here records a series of observations made on a subject "who both experiences hallucinations and can produce automatic (subconscious) script without awareness of what the hand is writing." In a first set of studies the subject wrote automatically regarding some topic suggested to her and at the same time indicated the moment of appearance and disappearance of any "hallucinations" which occurred during the writing. These hallucinations were described from memory, either at the time or subsequently. In a second series, hallucinations were induced by crystal gazing, this time without any suggestion as to topic, and while describing the visions the subject performed automatic writing. In both series "the method of subconscious introspection," answering questionnaires in automatic script, was employed to obtain details of the content of the subconscious during the time of the experiments.

The results show clearly that the conscious images (hallucinations) were correlated with, and followed the train of, subconscious thought. Furthermore, it was learned that an imagery occurred in the subconscious similar to that which accompanies conscious thought, though without awareness, and that this preceded in time the conscious hallucinations. In the second series of experiments, the script showed some striking misspellings and distortions, besides being so brief and symbolic as to show at first glance little connection with the vision. This was explained by the fact that the subject of the experiment had previously suffered from dissociation into a "dual personality." In the automatic writing, the full statement of the happenings in the subconscious was secured only by "tapping" the second personality system.

The principal conclusions reached from this series of careful records are that such dreamlike fantasies whenever encountered are "indications of a dissociated subconscious process and its content." "The genesis and psychopathology of the psychosis are to be found in the forces which have determined the dissociation and motivated the subconscious process."

SINGER, Chicago.

THE SIGNIFICANCE OF NYSTAGMUS IN A LESION OF THE ABDUCENS NUCLEUS. JOSHUA ROSETT, Neurol. Bull. **3**:332 (Sept.-Oct.) 1921.

On June 1, 1921, the patient, aged 52, awoke in the morning to find that his face had been drawn to the right side and that he could not close his left eye. He had double vision, which became worse in the course of the day, and which has persisted to date.

Except that he was a heavy drinker for a number of years, his history was negative. The interesting physical findings were: paralysis of the external rectus muscle of the left eye; inability to move the right eyeball to the left when a similar effort was made on the part of the left eye; rhythmical nystagmus of 1 mm. at the rate of four a second when the eyes were turned in a horizontal direction to the right; flaccid paralysis of the facial muscles of the left side; and definite loss of gustatory sense on the anterior two thirds of the left side of the tongue. About ten weeks later all these symptoms disappeared, with the exception of paralysis of the external rectus muscle of the left eye.

The author localizes the lesion around the sixth nerve nucleus, which, extending peripherally, would catch the seventh nerve fibers as they hook over the sixth. The nervus intermedius carrying the fibers of taste would be caught by the lesion extending a few millimeters out from the abducens nucleus as the former bends down to enter the nucleus solitarius. The fact that a complete lesion of any nucleus must cut off the pathways connecting it with all other structures accounts for the inability to move the right eye to the left in the effort of conjugate movement of both eyes, while the right eye can be turned inward in a movement of convergence. The author's theory in regard to nystagmus is that there is no connection between the vestibular nuclei and the nuclei of the oculogyric mechanism, as in the case of all the other motor nuclei of the central nervous system. The explanation of this is based on the fact that the center of gravity is not changed in the eyeballs on movement of the body, and that the adjusting of the visual axes to the body and head would be entirely between the optic mechanism and the cerebellum. Assuming this line of reasoning to be correct and the nystagmus in the case of the patient mentioned a mode of incoordination of the nature of dysmetria, the author assumes that there is an injured conduction pathway from the cerebellum to the nucleus of the sixth nerve, an interrupted rubro-abducental tract.

In the discussion of the author's case which followed, the opinion was given that the nystagmus was neither vestibular nor cerebellar, but a paretic ocular nystagmus due simply to imbalance in the muscles, entirely peripheral in nature.

STACK, Philadelphia.

AN EXPIATION PROCESS IN A CASE OF SCHIZOPHRENIA. HENRY DEVINE, J. Neurol. & Psychopath. **2**:224, 1921.

The report is of exceptional interest for the reason that the account was given spontaneously by the patient, without suggestion by the examiner. The analysis has been limited strictly to the facts and is admirably clear and connected. The chief features in the outward appearance of the patient were a wandering, shut-in existence extending over many years, stilted phraseology with many mannerisms and neologisms, an attitude of mocking humility and exaggerated politeness and episodes of extreme irritability and antagonism.

When secured, the detailed story revealed the presence of hallucinations, called by the patient "strengths," which referred to offenses actually committed against parents, friends and others during childhood. The mannerisms and attitudes of the patient were clearly stated to be expiatory penances which he was compelled to repeat innumerable times for these offenses and for any similar thought which he might entertain even for a moment of similar activities now. The "strengths" would trick him into such thoughts, often obscurely symbolic, in order to have the pleasure of punishing him.

Dr. Devine concludes that these indicate an ambivalent tendency toward sadism and masochism, using these terms, not in relation to specific sex urge of which the patient spoke hardly at all, but rather in the sense of a "more fundamental than even sex" instinct which he suggests corresponds with Schopenhauer's "will to live" or Nietzche's "will to power." The richness and fulness of intellect revealed by the patient in his descriptions are stressed as evidence against any true dementia, and the conclusion is reached that "the psychosis is the outcome of an arrest or fixation of the instinctive and emotional life at a primitive or infantile level," in spite of the high level of intelligence which has been reached.

SINGER, Chicago.

PAIN IN THREATENED AND REAL GANGRENE OF THE EXTREMITIES: ITS RELIEF. BERNHEIM, Am. J. M. Sc. **163**:517 (April) 1922.

The author does not attempt to suggest a cause for the intense pain in gangrene of the extremities. He says that he is at a loss to explain it. Pain, however, is an early and constant symptom of the disease, and first manifests itself as a numbness and tingling of the toes and feet, together with a sensation of cold and a marked blanching of the extremities. The patient usually first complains of pain in the extremity so severe that it wakens him at night. The condition is often mistaken for a neuritis, whereas the circulatory system is at fault. There is no nerve degeneration. Relieving the gangrene relieves the pain, and measures are instituted for this purpose.

The pain is usually so intense that huge doses of sedatives are required, the most efficient being sodium bromid in cautious doses. Rest is important. Blood vessel exercise is advocated by plunging the leg first in a pail of ice water and then after a minute into a pail of very hot water for a few seconds. The process is kept up for five minutes, and finally a prolonged bath in the hot water brings vascular dilatation. This is repeated three times a day if the patient can stand the procedure. Following the hot and cold plunges, the member is dried and rubbed with olive oil. An electric vibrator is then used similar to that used on the face in barber shops. This is applied for ten minutes at a time for several applications daily.

One liter of Ringer's solution is given into the duodenum by tube each day until the patient becomes unable to take the treatment.

The Moskowicz method of testing the reaction time of the vessels is used once a day. The member is kept clean and all precautions taken to prevent ulceration. Strict palliative measures are suggested. Amputation is to be the last resort. Arteriovenous anastomosis has not proved satisfactory. The Leriche operation for stripping the vessels has not been tried by the author; he feels that it requires more confirmation.

When operation is resorted to, the limb should be amputated high enough to ensure enough blood for healing of the flap. This the author has found occurs only when the member is taken off above the knee. Amputations at lower levels have to be repeated.

TEMPLE FAY, Philadelphia.

A STUDY OF THE PHYSIOLOGY OF THE NERVOUS SYSTEM OF INSECTS. II. RESEARCH ON CIRCUS MOVEMENTS PROVOKED IN COLEOPTERA. EDGARDO BALDI, J. Exper. Zool. **36**:211, 1922.

Injury to one side of the supra-esophageal ganglion of certain beetles brings about a change in behavior which is in harmony with the chiasmatic

arrangement of the fibers running from the brain to the lower ganglions, not being restricted to one lateral half of the body but involving the whole organism. This is especially clear in the antimere opposite to the lesion. This injury probably provokes a general increase of movement which is manifest in the appendages of the injured side. The final effect is a disturbance of equilibrium in the normal functioning of the musculature, in which the activity of the flexor muscles of the uninjured side predominates. Thus movement in a circle is produced. The movement in a circle is due for the most part to the traction predominatingly exercised by the first two appendages of the uninjured side, this traction being variously directed with respect to the sagittal plane. It is helped by the propulsive arcuate movements characteristic of all the appendages of the injured side. That the movements of flexion are the principle cause of circus movements is proved:

(*a*) By the impossibility of eliciting such movements by the artificial exaggeration of the propulsive activity of one side.

(*b*) By the reestablishment of straight locomotion, when, by amputation of leg segments, the movements of traction are rendered impossible.

(*c*) By the persistence of circus movements where only the propulsive arcuate movements are prevented in the legs of the injured side. Thus circus movement results from an alteration of the general conditions of symmetry of the organism, but from such an alteration as affects the entire organism, assuming various aspects in different regions.

WYMAN, Boston.

A CASE OF VENOUS ANGIOMA OF THE CEREBRAL CORTEX.

H. CAMPBELL and C. BALLANCE, Lancet **1**:10 (Jan. 7) 1922.

These authors report the case of a man, aged 23 years, who at 13 first noticed weakness in the left arm and leg, which increased progressively. At about the same age, he developed seizures which began with numbness in the left great toe and extended upward. When the numbness reached the left hand, it closed; and when the left temporal region was reached, the head was drawn to the left and the mouth opened. These seizures were followed by severe headaches. Until three months prior to admission (Nov. 1, 1920) the seizures occurred every six months; but they became more frequent and were accompanied by clonic spasms. Examination on admission showed the presence of a mild degree of left hemiplegia with increased deep tendon reflexes on that side, and loss of the left abdominal reflex. Ankle clonus was present on the left side, but no Babinski's sign could be elicited. There was diminished sensory discrimination over the left foot, leg, arm and forearm. The optic disks were normal. A diagnosis of a progressive neoplasm involving the right precentral and postcentral convolutions was made. At operation, April 2 and 9, 1921, an extensive venous angioma covering the external and mesial surface of the right cerebral hemisphere was found. The nevus grew from the vessels of the pia and cortex and could not be removed. Six months after operation, he had had only two convulsive seizures. Campbell and Ballance believe that the long history (ten years), the slowly progressive left-sided paresis, the absence of optic neuritis, seizures commencing with a sensory aura of the left great toe, failure to localize light touch on the skin of the left foot, leg, hand and forearm, point to the presence of a widespread chronic cortical lesion.

POTTER, Mercer, Pa.

A CASE OF PROGRESSIVE MUSCULAR DYSTROPHY. E. D. FRIEDMAN, J. Nerv. & Ment. Dis. **54**:294 (Oct.) 1921.

The patient, a schoolboy of 16 years, complained of weakness of both legs for fifteen years. His family history was negative. He began to walk at 9 months of age and walked for one month. His legs then became weak, and he crawled until he was 5 years old. After that he was able to stand again, but he noticed weakness of his legs which has persisted. Physical examination showed an attitude and gait characteristic of muscular dystrophy. The patient had a waddling gait and a marked lordosis and arose from the floor with difficulty. The muscles involved were: the sternomastoid on the left, the right spinati, the deltoids, the left pectorals, the left biceps, and the erector spinae. Both quadriceps muscles were weak, with little muscle tissue remaining. The adductors of the thigh were involved as were also the dorsiflexors of the feet. Both knee reflexes were absent. Disturbance in the glands of internal secretion was shown by an overgrowth of hair at the bridge of the nose, the presence of large hands and feet, and female distribution of pubic hair. All laboratory reports were negative. There was no response to galvanism or faradism in the quadriceps muscles.

That the case belongs in the group of muscular dystrophies is evidenced by the combination of atrophy and hypertrophy and the absence of both fibrillation and reaction of degeneration; likewise, to some extent by involvement of the proximal group. The author believes that the lesion is at the neuromuscular junction, because there is no definite symmetry in the localization of the lesion. He thinks this might explain the absence of knee jerks in this case. He also believes that all cases in which no pathologic condition can be demonstrated in the anterior horn cells or their axons should be grouped under the term muscular dystrophy.

The pathologic picture of this disease is usually that of a primary muscular atrophy. There is atrophy and hypertrophy of the muscle bundles with increase in the number of nuclei, proliferation of the perimysium, a deposit of fat cells in the muscles and vacuolization of the muscle cells. Erb looks on hypertrophy as a preliminary stage of the disease.

The prognosis is good unless the muscles of respiration are involved. The disease may last from thirty to forty years.

STACK, Philadelphia.

CONCERNING DIFFUSE GLIA REACTIONS. J. L. PINES, Schweiz. Arch. Neurol. u. Psychiat. **11**:112, 1922.

A number of authors, including Henneberg, believe that the normal glia cell may be transformed into a glia tumor cell and that no sharp division is possible between a diffuse glioma and a secondary reactive glia proliferation; also that the proliferation does not start from a focus of glia cells, but is diffuse from the outset. Ceni and Chiari share this belief. Stumpf believes that tumor cells of this type proliferate in the pre-existing neuroglial syncytium, the irritant bringing about a progressive proliferation of glia cells. Similar conclusions were drawn by Landau, who assumes a rather general congenital abnormality. The author believes that the increase in glia cells in his case is reactive and gliomatous in character.

As irritants, Merzbacher includes tumor, hemorrhage and softening; however, these factors do not seem sufficient in themselves to result in true glioma formation. According to most authors, these determinants reside in the glia

cells themselves, which, according to Cohnheim, as is well known, represent displaced embryonic cells. Lenhossek suggests agenetic, or incompletely developed glia cells which have not been implanted from the outside, but which retain their embryonic character and fail to differentiate fully. Exactly what is required for the production of diffuse reactive glial proliferation approaching the tumor type of cell, in contradistinction to the normal glial cell reaction, is not yet known. The organs of internal secretion, such as the choroid plexus, have been looked on as supplying the required stimulant. Several authors have laid great stress on the association of glioma with other constitutional anomalies. Anton called attention to a case showing diseased suprarenals and hypertrophy of the brain and persisting thymus. Neumann reported a case of glioma, cyst of the pineal body, persistent thymus and enlarged thyroid. Bartel reported a series of gliomas associated with status thymicolymphaticus. Pines believes that this is more than coincidence.

In conclusion, he believes that there was present in his case an individual factor expressing itself in heightened glia reaction, the precipitating moment being furnished by the irritation resulting from the primary dural tumor.

WOLTMAN, Rochester, Minn.

CASE OF SPINAL SPASTIC PARALYSIS (PRIMARY LATERAL SCLEROSIS). J. T. BROADWIN, J. Nerv. & Ment. Dis. **54**:289 (Oct.) 1921.

A man, 29 years of age, complained of stiffness in both lower extremities, which began at about 12 or 13 years of age, progressed rather slowly up to three years ago, and has remained stationary since then. The family history was negative. At 16 the patient noticed that he wore out the inner sides of the soles of his shoes rapidly. He was unable to state which leg was first involved. There were no subjective sensory symptoms other than occasional transient pains in his feet.

The interesting physical findings were: coarse nystagmoid movements with a rotary element in both eyes on fixation to extreme lateral planes; a fine tremor of the fingers of the outstretched hands; spastic gait, the right leg appearing more spastic; the legs moving en masse as though walking on stilts, with a scraping of the feet. Considerable resistance was encountered during passive movements of both lower extremities, more marked on the right. The gross motor power was well preserved. There were no atrophies or fibrillary tremors, and likewise no objective sensory findings. There was a slight lordosis in the lumbar region, a roentgenogram of which revealed no abnormal findings. The reflexes of the upper extremities were lively; the knee and ankle reflexes were exaggerated; patellar and ankle clonus and a positive Babinski sign were present.

Oppenheim states that in the great majority of cases that first appear to be a spastic spinal paralysis, symptoms later appear which show that behind this picture there frequently lurks some other disease of the nervous system, such as disseminated sclerosis, chronic myelitis, compression of the cord and occasionally amyotrophic lateral sclerosis or cerebral disease. Necropsy findings of this disease have formerly been reported by Erb, Spiller and Strümpell. The course of the disease is slow, and it may even cease to progress.

In the treatment of severe forms, division of certain of the posterior lumbar roots is sometimes advised.

STACK, Philadelphia.

AN ANGIOMA OF THE PONS. LEYSER, Monatsch. f. Psychiat. u. Neurol. **51**:83 (Feb.) 1922.

Angioma of the pons is a rare condition. Only seven cases are reported in the literature. The author's patient was a woman, aged 20, always well up to the present illness. The onset of the illness was sudden. She cried out, fell to the ground, and had a convulsive seizure. After half an hour, there was deep unconsciousness. The pulse was slow, respiration irregular; there was vomiting, and incontinence of urine. The pupils were small, equal and rigid. The left corneal reflex was gone. Jerking movements of the left arm and leg and flaccid paralysis of the right arm and leg were present. On the fourth day, there was a beginning left facial paralysis, which increased to complete paralysis in a few days. There was a skew deviation of the eyes, the left eye pointing down and in, the right eye up and out. With the exception of a slight clearing on the tenth day, the patient remained deeply unconscious to the end. Death occurred on the twentieth day.

Necropsy revealed a small left-sided angioma of the pons, with an extensive hemorrhage which had caused much tissue destruction, and had broken into the ventricle.

The most striking feature was the so-called Hertwig-Magendi symptom, or skew deviation of the eyes. The eye on the affected side turned down and in, the eye on the opposite side, up and out. The pathologic basis for this is not definitely known. Some consider it due to a lesion of the middle cerebellar peduncle. Biehl believes it to be due to an affection of the vestibular nucleus. The author attributes the symptom in his case to the destruction of the left vestibular nucleus.

The combination of this left-sided skew deviation with a right-sided hemiplegia forms a type of hemiplegia alternans hitherto undescribed in the literature. The author suggests as a name "hemiplegia alternans vestibularis." The syndrome will probably never be found pure, but associated with involvement of the homolateral abducens and facial nerves.

SELLING, Portland, Ore.

RECURRENT HYPERTROPHIC NEURITIS. F. J. NATTRASS, J. Neurol. & Psychopath. **2**:159, 1921.

A remarkable case, apparently unique in the literature, is grouped by the author with the "hypertrophic interstitial neuritis" of Déjerine and Sottas in spite of marked differences, notably the recurrences. The patient had suffered two apparently similar attacks, which had been regarded as anterior poliomyelitis. The first occurred at the age of 4, with recovery in six months, and the second at the age of 17, with recovery in three months. The third attack began suddenly at the age of 18, was afebrile and progressed rapidly to almost complete paralysis, accompanied by pains, numbness and tingling, in two weeks before the patient came under observation. He then presented flaccid paralysis of the arms, most marked in the distal segments, with atrophy and electrical changes which in the hands constituted a complete reaction of degeneration. The reflexes were absent, and there was tremor on movement with well marked adiadokokinesis. The trunk muscles were weak, but the abdominal reflexes were present.

In the lower extremities, only slight movements were possible at the hip and knee; below, paralysis was complete with atrophy and reaction of degeneration. The reflexes were absent.

The cranial nerves were not involved, and there was no loss of sphincter control. Objective sensory disturbances, with the possible exception of defective pallesthesia, were absent. The muscles were tender everywhere, even in the cranial nerve group. The large nerve trunks (not the cutaneous nerves), where palpable, were uniformly swollen, hard and tender.

Improvement began within a day or two after admission to the hospital, and at the end of three months functional recovery was practically complete, although knee and ankle jerks were still absent, and the electrical reactions were not normal.

SINGER, Chicago.

EPILEPTOID OR FAINTING ATTACKS IN HYPOPITUITARISM. L. PIERCE CLARK, Am. J. M. Sc. **163**:211 (Feb.) 1922.

The occurrence of fainting attacks and epileptoid states in hypopituitarism are considered, and an attempt made to show the relationship between them, clinically and therapeutically.

The seizures usually occur in rapidly growing adolescents who are subject to relatively benign fainting attacks, which may at first simulate larval forms of petit mal epilepsy. They are to be differentiated from the latter condition by the absence of the epileptic character and the general physical and mental stigma of the disorder. The syncopal states are only a part of the obscure clinical picture of dyspituitarism, in which the author states there appears to be an excessive functioning of the anterior lobes of the pituitary gland.

Coincident with the fainting attacks there may be low blood pressure, slow pulse, vasomotor ataxia and a host of muscular and skeletal defects in development. The cause is suggested in the irregular structural growth of the body, resulting in a disharmony of function of the vasomotor and sympathetic systems. It is also pointed out that in the physical sphere one may encounter not infrequently character delinquincies and slow mental development.

The line of corrective treatment advised is outdoor life, exercise and an abundance of sleep plus the administration of specific glandular substance. The author says that patients recover spontaneously after rest and leading an outdoor life. Three cases are cited—in one posterior pituitary and thyroid medication was prescribed, in the other two, more extensive rest. All patients recovered normal balance. The use of glandular therapy is advised in a rational and cautious manner.

TEMPLE FAY, Philadelphia.

ON THE PRODUCTION OF NEURO-MUSCULAR PATTERNS BY THE RELEASE OF SPINAL INTEGRATIONS AFTER DECEREBRATION. W. M. KRAUS and A. M. RABINER, J. Neurol. & Psychopath. **3**:209, 1922.

Developmentally, the muscles and nerves may be divided into dorsal and ventral groups. Analysis of the postures of decerebrate rigidity and of the flexion stepping and arm reflex according to the particular groups of muscles involved reveals the following: In decerebrate rigidity the neuromuscular apparatus in action is dorsal for the body axis (trunk and neck), ventral at the shoulder or hip, dorsal at the elbow or knee and ventral at the wrist or ankle and parts below. The neuromuscular pattern for axis and appendages can therefore be expressed by the formula DVDV. In the flexion reflex posture exactly the reverse arrangement is found, VDVD.

A series of cases, three of lethargic encephalitis and one each of a septic meningo-encephalo-myelitis and extradural spinal tumor, are studied and analyzed in this manner, and the essential similarity demonstrated. The encephalitic cases were all of the choreic and kinetic type, and in two of them besides the DVDV action which was common to all three, there were occasional movements of the VDVD pattern. In the two remaining cases, the pattern was fragmentary but true to form.

In a further discussion, it is concluded that the integrations for these patterns must be spinal and that they may be activated by two forces, one kinetic and the other static or tonic, but that the muscles are activated in clearly defined patterns.

SINGER, Chicago.

CALCIFICATION OF THE PITUITARY WITH HYPOPITUITARISM AND WITH SYMPTOMATIC TREATMENT. PFAHLER and PITFIELD, Am. J. Med. Sc. **163**:491 (April) 1922.

The roentgen-ray findings in nine cases are presented, with the results of treatment and the use of glandular therapy.

There were varying degrees of calcareous deposit in and about the pituitary region, and the clinical pictures of the cases were surprisingly similar and sufficiently clear to draw attention to the dysfunction of the gland. Fatigue, depression, vagotonia, headache, lethargy, high sugar tolerance, low blood pressure, vasomotor instability, rhinorrhea, belching of gas and absence of nail crescents were the symptoms most often noted.

The article is well illustrated with plates of skilfully made roentgenograms. Four of the cases were found between the ages of 19 and 27, the remaining five between the ages of 38 and 54. In all but one of the cases there were definite symptoms that pointed to pituitary disorder. In the one exception, while there was no definite evidence of pituitary disease, the vagotonia and neurasthenia improved under pituitary feeding. The roentgen ray showed a thickening of the capsule surrounding the gland rather than an infiltration of salts into the substance itself. The authors feel that the presence of such infiltration within the gland is a definite indication of disease either present or past, though in the other cases noted the gland seemed more dense than normally, without any signs of glandular disturbance.

The treatment instituted was the administration of whole gland pituitary in doses as high as 6 gm. a day. Many patients apparently were markedly improved by this treatment. The authors advise the use of stereoscopic plates when investigating disorders of the pituitary.

TEMPLE FAY, Philadelphia.

STUDIES IN ASYMPTOMATIC NEUROSYPHILIS. IV. THE APPARENT ROLE OF IMMUNITY IN THE GENESIS OF NEUROSYPHILIS. ALBERT KEIDEL, J. A. M. A. **79**:874 (Sept. 9) 1922.

Investigations show that neural invasion occurs in probably all cases of early syphilis, but clinically less than 50 per cent. of these patients are neurosyphilitic. In the group studied by the author, he found that 21 per cent. of all patients with late syphilis, including cases of latent syphilis, present definite evidence of clinical neurosyphilis; 21.6 per cent. of patients with late syphilis with apparently normal nervous systems have definite abnormalities in their cerebrospinal fluid showing late asymptomatic neurosyphilis, making a total of 42.6 per cent. of all patients with late acquired syphilis who are

actively neurosyphilitic. This represents the percentage of patients with late syphilis who failed to acquire an immunity sufficient to suppress the neurologic invasion. An additional 17.7 per cent. of all those with late syphilis show suggestive evidence of immune protection. The remaining 39.7 per cent. are patients with apparently normal cerebrospinal fluids, in whom neurolatency has developed without demonstrable neurologic damage, or who had developed protection before damage was done. The author concludes that the character of the early tissue reaction to the syphilitic infection, the intensity, continuity and duration of the therapeutic attack, and the occurrence of pregnancy in women, markedly modify the incidence of neurosyphilis, both clinical and asymptomatic.

NIXON, San Francisco.

ON FROIN'S SYNDROME AND ITS RELATION TO ALLIED CONDITIONS IN THE CEREBROSPINAL FLUID. J. G. GREENFIELD, J. Neurol. & Psychopath. **2**:105, 1921.

After a brief review of the literature, three cases presenting the complete syndrome and eighteen a partial reaction are outlined. Under this last category are included fluids with a high albuminous content but in which the yellow color may not be marked, or in which there is no spontaneous clotting owing to the absence of fibrin ferment. Addition of the latter will cause clotting. The complete reaction was observed in Paget's disease of the spine, carcinoma of the spine and intradural myxoma. The partial syndrome occurred in cases of acute myelitis, staphylococcus meningitis, cerebrospinal syphilis, thoracic aneurysm pressing on the cord, Pott's disease and spinal tumor. Froin's syndrome is also recorded in the literature as occurring in polyneuritis and Landry's disease.

The author reviews and discusses the theories which have been suggested to explain the condition; and, after pointing out that the fluid approximates in character toward that of the blood serum, concludes that this is brought about by blocking off the fluid in the lumbar sac from that in the spinal and cranial cavities above. The degree of approximation to serum depends on the completeness of the block, and its occurrence is aided by two factors: venous congestion or inflammation with resultant transudation and the entrance of lymph into the subarachnoid space by way of the perineural or perivascular lymphatics. The latter factor explains the occurrence of the syndrome in polyneuritis.

SINGER, Chicago.

THE VALUE OF THE ROUTINE USE OF THE COLLOIDAL GOLD REACTION IN ACUTE EPIDEMIC POLIOMYELITIS. REGAN and CHENEY, Am. J. Dis. Child. **23**:107 (Feb.) 1922.

The authors found a distinct diagnostic value in the use of the colloidal gold reaction in their series of twenty-one cases. The curve appeared in all cases, and fell in a definite portion of the reaction zone.

The curve shows a slight rise in the early syphilitic area. It is expressed numerically as 1 1 2 2 1 0 0 0 0 0. The curve lies in the same zone at all periods of the disease. It is highest at the onset of symptoms and therefore has a diagnostic value. There is a gradual fall until the eighth week, but it may persist in cases in which there is a residual paralysis or polyneuritis. A chronic case with recurrent attacks showed a typical curve for five years after the initial onset.

As the curve is similar to a weak tabetic reaction, the history, age and Wassermann reaction must rule out syphilis. Meningismus gives no reaction, so that the use of this test would be of value in differentiating poliomyelitis from that condition. Epidemic encephalitis gives such variable curves that it is easily distinguished from those falling in characteristic zones. In cases in which the curve is prolonged into the higher dilutions (known as the meningitic zone), tuberculous meningitis must be ruled out, otherwise the curves are quite distinct.

The authors found the reaction in all cases up to the fourth week of the disease. It subsided with the acute stage and the gradual improvement of the patient.

TEMPLE FAY, Philadelphia.

THE ARGYLL ROBERTSON PUPIL. S. A. K. WILSON, J. Neurol. & Psychopath. **2**:1, 1921.

The Argyll Robertson pupil is not necessarily accompanied by myosis and may be due to lesions other than those of syphilis. The centripetal path for the light reflex is physiologically, and probably also anatomically, distinct from that subserving light perception. The reflex fibers leave the optic tract before the external geniculate body is reached and pass to the superior colliculus by way of the brachium, although the exact path is not known. Partial decussation takes place in the chiasm and again on leaving the colliculus. The convergence and accommodation reaction of the pupil is an associated movement and is activated from the cortex, though the center is not known; and the final infranuclear path is the same as that for the light reflex.

A series of clinical observations is reported to demonstrate the occurrence of an Argyll Robertson pupil in epidemic encephalitis, disseminate sclerosis, tumors in the region of the third ventricle and aqueduct, and syringobulbia. It may also arise from injury to the bulbus oculi and in chronic alcoholism. The most common site of the lesion is in the neighborhood of the aqueduct, but the syndrome may arise from damage to the optic nerve or tract distal to the geniculate bodies. It is suggested that the damage is most often (syphilis and disseminate sclerosis) an ependymitis or subependymitis due to a toxi-infective, lymphatic invasion from the cerebrospinal fluid.

SINGER, Chicago.

ORGANIZATION OF SOCIAL WORK IN A STATE HOSPITAL. MORTIMER W. RAYNOR, State Hosp. Quart. **7**: No. 3 (May) 1922.

Raynor gives the organization of social work in the Manhattan State Hospital in the following stages:

1. At time of admission of patients:
   - (*a*) Investigations to locate interested friends and relatives
   - (*b*) Amelioration of home and family conditions
   - (*c*) Gathering of social history data
2. Follow-up work during hospital residence:
   - (*a*) Amelioration of home and family conditions
   - (*b*) Development of plans for leaving hospital
3. Preparole:
   - (*a*) Investigations and establishing entrée with family.
   - (*b*) Report to physician
   - (*c*) Adjusting of home and family conditions for the patient's return home
   - (*d*) Securing of home or boarding house and a job

4. After-care:
   (*a*) Supervision through clinics
   (*b*) Supervision through visits to home and plan of employment
   (*c*) Advice and assistance in maintaining adjustments and promoting a salutory atmosphere in the home
5. Mental hygiene in community:
   (*a*) Contact with other agencies doing social work, the schools, and correctional institutions and agencies, etc.

The need of close cooperation and frequent conference between the physician and social worker is clearly stated in this article.

EBAUGH, Philadelphia.

BILIOUS ATTACKS AND EPILEPSY. C. W. VINING, Lancet **1**:122 (Jan. 21) 1922.

This author calls attention to the association of bilious attacks and epileptic seizures. In a series of 194 cases in which accurate information was obtainable, seventy-eight patients gave a history of bilious attacks, and ten a history of periodic headaches. In forty-eight cases, these phenomena continued in association with the epilepsy; in twenty cases, they preceded the epilepsy and ceased with the onset of the seizures. Vining, in an attempt to combat the critics of his theory, elicited information regarding bilious attacks from 100 persons attending the public dispensary for conditions other than epilepsy. In this group, twenty-one suffered with bilious attacks, but three of them gave a definite history of epileptic seizures, and eight such a history in near relatives. He also notes a close relationship between nocturnal enuresis and epileptic seizures. The author concludes that some cases, especially in children and adolescents with a history of bilious attacks, associated with headaches or nocturnal enuresis, should be considered as potentially epileptic, and suitable steps should be taken when possible to prevent the development of the convulsive habit.

POTTER, Mercer, Pa.

THE CRAWLING OF YOUNG LOGGERHEAD TURTLES TOWARD THE SEA. G. H. PARKER, J. Exper. Zool. **36**:323, 1922.

By placing newly hatched loggerhead turtles in a great variety of environmental conditions and watching their behavior, the following conclusions were reached:

Newly hatched loggerhead turtles find their way from their nests to the sea in consequence of at least three factors: first, their positive geotropism as shown in their tendencies to move down slopes; second, their response to their retinal images in that they move toward regions in which the horizon is open and clear, and away from those in which it is interrupted, and third, their probable response to color in that they move toward blue areas rather than toward those of other colors (Hooker). These animals are not appropriately described as either negatively or positively phototropic, but are to be regarded as exhibiting a more complex condition, in that they respond to the details of their retinal images rather than to these images as a whole.

WYMAN, Boston.

THE CEREBROSPINAL FLUID; ITS SOURCE, DISTRIBUTION AND CIRCULATION. R. M. STEWART, J. Neurol. & Psychopath. **3**:144, 1922.

This is a critical review of the literature, and it reaches the conclusions that the only features which appear to be definitely established are: 1. The

fluid is derived chiefly from the choroid plexus and in a subsidiary manner from the perivascular spaces and possibly the membranous surfaces of the brain. 2. Absorption in the brain occurs chiefly through the arachnoid villi and to a lesser extent through the lymph sheaths of the cranial nerves. In the spinal cord elimination takes place chiefly through the lymphatic system. 3. Fluid circulates in the brain ventricles and subarachnoid spaces, but to a limited degree in the spinal spaces.

It cannot be definitely stated whether the fluid is a secretion or a dialysate, the rate of production and absorption, or whether the fluid serves as a lymph or circulates within the brain substance.

SINGER, Chicago.

WAR PSYCHOSES, THE INFECTIVE EXHAUSTIVE GROUP. D. K. HENDERSON, Glasgow M. J. **96**:321 (Dec.) 1921.

This author reports the study of a series of 115 cases in patients invalided from Mesopotamia, Salonica, India and Egypt. Malaria is given as the cause in forty-four cases, dysentary in 12, enteric fever in 5, heat exhaustion in 13, influenza in 7, septic wounds in 5, and miscellaneous and mixed causes in the remaining 29 cases. Of these patients, 13 were dull, apathetic and depressed; 19 showed hallucinations of a depressive character; 26 were irritable, suspicious, delusional and hallucinatory; 42 were delirious; 7 wandering and amnesic; 3 stuperous; 2 were maniacal, and 3 showed a rather characteristic Korsakoff's syndrome. Henderson concludes that exhaustion as a primary factor in the causation of mental illness has been greatly overrated, although it is of importance as a contributory factor. The symptom picture in infective-exhaustive psychoses varies, and depends chiefly on the innate characteristics of the person.

POTTER, Mercer, Pa.

THREE CASES OF MANGANESE POISONING. J. R. CHARLES, J. Neurol. & Psychopath. **3**:262, 1922.

Of the three cases, two were at first diagnosed as cases of functional disorder, the third as syphilitic myelitis. The diagnosis of manganese poisoning was finally made because all three patients had worked in the same factory in an atmosphere of manganese dust. All presented more or less aphonia, mask-like facies, weakness in the arms and legs with marked spasticity in the legs. Sensation and coordination were unimpaired. The tendon reflexes were brisk, but there was no clonus, and the plantar reflexes were of flexor type. There was no dementia. The shortest duration since removal from the manganese atmosphere was two years, but there was no marked improvement. The damage was therefore considered to be permanent and in all probability located in the region of the basal ganglions.

SINGER, Chicago.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Ninety-Eighth Regular Meeting, Oct. 3, 1922

FOSTER KENNEDY, M.D., *Presiding*

DOUBLE PAPILLEDEMA CAUSED BY BLOCKING OF CORD AT FOURTH CERVICAL VERTEBRA GREATLY RELIEVED BY OPERATION. DR. T. K. DAVIS.

A man, aged 24, with a negative family and personal history, for two years before admission was ill with headache, dizziness and vomiting; he did not have diplopia or fever. He improved and returned to work (clerical), and was well for a year. The headaches then returned, and the legs and arms felt weak. On admission to Bellevue Hospital, in February, 1922, examination revealed slight papilledema diminution of the deep reflexes in the arms, normal reflexes in the legs, a Babinski sign on the left side and normal abdominal reflexes; there were no sensory changes in the upper extremities. A roentgenogram of the spinal column showed no bone involvement or spondylitis, and the roentgenogram of the skull was negative. The spinal fluid was negative for cells and for colloidal gold. Both the spinal fluid and blood Wassermann tests were negative. There was no cervical rib. The youth showed some congenital abnormalities: hypospadias, one undescended testicle, and a doubling up and over of the fifth toe on each foot.

The headaches were greatly improved by lumbar puncture. The patient was kept under observation. Two months later he returned. He had weakness of the arms and hands, marked atrophy of the arms, hands and shoulder girdles and increased headaches and papilledema. The triceps reflexes were now absent; the biceps reflexes were present though weak. There was hypesthesia in the distribution of the third to eighth cervical, and first thoracic roots inclusive. Over the first dorsal vertebra there was also a disproportionate disturbance of temperature sense. On one occasion he had a general convulsion without loss of consciousness, lasting only two minutes. During this there were clonic spasms of all extremities, as well as lateral rotation of the head and eyes from side to side. In view of this convulsion, the severity of the headaches and the increasing papilledema, a laminectomy was deemed advisable, and the third, fourth and fifth cervical laminae were removed in June, 1922. A tense dura was found, with the cord completely filling the dural cavity and an apparent lack of fluid below an enlargement at the fourth cervical segment. Exploration was made for a dural tumor, but none was found.

The swelling about the fourth cervical was incised in the posterior midline, and a cavity was disclosed, which on probing was found to be about 1½ inches (3.81 cm.) long. One interesting feature is that the operator for some reason, not fully explained, was unable to pass a probe upward through the foramen magnum. The patient made an excellent recovery, and the papilledema disappeared. Postoperative treatment has consisted of massage. The greatest disability remaining is in the left deltoid and left pectoral muscles, but even these muscles respond to both faradism and galvanism, and with normal polarity.

DISCUSSION

DR. FREDERICK TILNEY: Did the papilledema disappear after operation?

DR. T. K. DAVIS: The optic cups are still somewhat obscured, and the edges of the disks are a little irregular in outline. These are the only changes, and they probably indicate a certain degree of postneuritic atrophy.

DR. TILNEY: The case appears interesting because the change, so far as one knows, was so remote from the particular lesion. Certain things in the symptomatology, such as the convulsive seizure, would lead one to believe that the lesion in the cervical cord was not the only change in the central nervous system, so that the papilledema might be explained by something else than the blocking of the subarachnoid spaces. When stigmas are so numerous, there may be other lesions, such as embryonic rests or cyst formation in other parts.

DR. FOSTER KENNEDY: The patient seemed to fulfil all the conditions required by our theoretical conceptions. The cervical gliosis was definite, with motor segment atrophies. The segmental sensory disassociation proceeded rapidly, coinciding with intense headache and severe papilledema; this seemed to demonstrate the fact that the papilledema was due to acquired hydrocephalus, and operation corroborated this explanation. The cord filled the canal; there was no pulsation below the level of the bulge. On incision of the dorsal column, a large cavity was found. This upheld our diagnosis in every respect, because the incision of the syringomyelic cavity and cord decompression produced amelioration of the papilledema. That there might be other embryonic defects of the brain which produced the convulsion and papilledema can neither be proved nor disproved, but the facts as we know them are opposed to that conclusion. The convulsion was synchronous with a period of tremendous intracranial pressure, and relief of the spinal pressure produced relief of the intracranial symptoms. These cases of high cervical lesions producing intracranial effects were first recorded in 1902, but this is the first case I have seen.

GENESIS OF CEREBELLAR FUNCTIONS. DR. FREDERICK TILNEY.

This article appears in full on page 137 of this issue.

DISCUSSION

DR. FOSTER KENNEDY: Invertebrates have no cerebellum as we know it, but the postural coordination of insects is perfect. Dr. Tilney's definition of motion as a "fluid stream of postures" is a sound one, and clinicians would agree with that interpretation. Cerebellar lesions produce a decomposition of rhythm, the stream being broken into its component parts. I should like to ask about the idea of the development of lateral portions in birds. I think in some birds, for example, the fighting ostrich and the hawk, there are seen distinct neokinetic powers in the independent action of one limb. Here are highly coordinated movements with almost no development of the lateral lobes of the cerebellum.

DR. I. STRAUSS: There is no doubt about the facts as set forth by the speaker in this painstaking work. Dr. Tilney's thesis involves the discussion of a philosophical question. Does function change morphology, or is morphology altered by something else? The old Darwinian theory of the survival of the fittest has had to be modified. There are myriads of mutations of species. The Darwinian law determines which of the variables should survive; but is it not more correct to say that there have been variables and that certain of these which have been found of use to the organism have been allowed to remain?

That would explain the development of the lateral lobes. In regard to the birds, many of their movements are independent. They are not all associated or automatic. The scratching of hens and the wing movements of some birds in fighting are independent. In the jump from birds to mammals there is considerable lateral lobe development, and there seems to be a gap in the line of reasoning which must be bridged over. Possibly there may be some other factor or force which has to do with development of this part of the brain.

Dr. I. Abrahamson: Decerebrate rigidity must be regarded as posture; removal of the cerebellum does not change it; removal of a portion of the brain-stem, especially in the neighborhood of Deiters' nucleus, produces loss of hypertonicity, a change from extensor to flexor postures. This region of the brain-stem is represented in part by the cerebellum bulbare (Tilney) which controls the trunkal muscles, but not the extremities; yet removal of this part causes marked change in the posture of the extremities. I believe that the function of the cerebellum is to regulate or synergize the movements in the passage from one posture to another, that it exerts little or no influence on posture itself.

Dr. F. H. Pike: Sherrington investigated decerebrate rigidity long before the work of Magnus. Decerebrate rigidity appears after transection of the brain-stem in front of the corpora quadrigemina, but the whole midbrain region must be left intact. The rigidity disappears when the dorsal roots of the spinal nerves are divided or when the dorsal and ventral spinocerebellar tracts are sectioned, with the dorsal roots intact. Transection of the brain-stem at the lower border of the midbrain also abolishes decerebrate rigidity. Ninian Bruce traced fibers originating in cells in the nucleus dorsalis (Clarke) up to the anterior and posterior corpora quadrigemina. Thiele had shown, some years before, that careful ablation of the cerebellum did not abolish decerebrate rigidity.

Dr. Frederick Tilney: I think there is a definite degree of neokinesis in the bird, which is associated with the incipient lateral lobe formation. There is, however, considerable work to be done before confirming this view. I think posture is an essential of motion. With reference to the philosophic criticism of Dr. Strauss, I think it would take us a long time to reach a definite conclusion, and the discussion would become purely an academic one. With reference to the priority of function or form, we must interpret facts as a form-function relation. I believe that form precedes function, because in passing from one form to another the alteration of an essential part alters the relations of all parts of the body. I cannot answer the question about invertebrates, as I have not made a study of this side of the work. I think we are beginning too high up in our neurologic studies, and shall have to look for the answer to some of our problems in the invertebrates. I am told by those who have studied the subject that there are parts in the anthropods and arachnoids which show cell groupings in communication with the peripheral organs and the semicircular canals, the proprioceptive system and corresponding organs. As far as decerebrate rigidity is concerned, I do not know what it is; no one does. It has not been analyzed.

DEMONSTRATION OF ANIMALS IN WHICH THE RESULTS OF RECENT LESIONS OF THE CEREBELLAR CORTEX OF ONE SIDE WERE CONTRASTED WITH THOSE RESULTING FROM EXTIRPATION OF THE OTIC LABYRINTH OF THE SAME SIDE.

Dr. F. H. Pike (by invitation).

In the cat, the postural disturbance was marked. The head was turned about the long axis of the body, from 45 to 90 degrees to the side of the lesion. If

the animal lay on the right or sound side, the tip of the nose was turned up to an angle of nearly 90 degrees with the table. When the animal attempted to walk, it rolled over repeatedly to the side of the lesion. There was no actual disturbance of the movements of the limbs, but the animal was unable to control its posture or position. It has been shown in other experiments that the head straightens up after division of the dorsal roots of the cervical nerves on the opposite side to that of the vestibular lesions. There are also rhythmical movements of the eyes, with a slow deviation to the side of the vestibular lesion, and a quick movement in the opposite direction.

It was formerly stated in the textbooks that the effects of cerebellar lesions were essentially similar to the effects of vestibular lesions. This was something that everyone knew, and the question was not open to argument. But in the animals (cats) exhibited, those with cerebellar lesions did not show any true ocular nystagmus. Slight oscillations or tremors of the eyes could be seen when the animal looked intently at an object near it. There was no torsion of the head, and the animal did not roll over when it attempted to walk. It turned to the side opposite to the lesion in walking, or went straight ahead. It could turn to the side of the lesion in walking. The fore foot on the side of the lesion was raised higher in stepping than the other fore foot. The symptoms observable were concerned with movement rather than with posture. Our observations on ocular movements in cerebellar lesions are in general agreement with those of Eckhard. He removed the cerebellum of a frog with extreme care, and, in those experiments in which he was positive that he did not injure the medulla oblongata, he did not get ocular nystagmus.

The posture of the pigeon with a severe cerebellar lesion is more affected than is that of the cat with a cortical lesion. There was some rigidity of the legs and toes in the bird exhibited, but this was not indicative of a cerebral lesion, and could not be called decerebrate rigidity. True decerebrate rigidity has not been described in the pigeon. It will be remembered, also, that in Hughlings Jackson's original figure of a child with a cerebellar lesion there was opisthotonos and extension of the limbs.

NEW YORK NEUROLOGICAL SOCIETY AND NEUROLOGICAL SECTION OF THE ACADEMY OF MEDICINE

Joint Meeting, Nov. 14, 1922

FOSTER KENNEDY, M.D., and S. PHILIP GOODHART, M.D., *President*

A COMPARATIVE STUDY OF THE ICEBOX AND WATER BATH FIXATION METHODS IN THE WASSERMANN REACTION AS APPLIED TO NERVOUS DISEASES. DR. LEWIS STEVENSON (by invitation).

The impression has gained ground among laboratory workers that the icebox method has given positive results in syphilis which have not been found with the older methods. Studies of 459 cases of syphilis in all stages made by Wile and Hasley in 1919, showed that the icebox method was more sensitive. Other workers confirmed this view. In the author's work on the blood Wassermann reaction in tabes, which by the older method is usually negative, the icebox method is found more delicate and reliable. It is especially applicable to syphilis of the nervous system, in which the percentage of positive

reactions has been found to be increased about 40 per cent. Perhaps in other forms of syphilis this would not be so marked. It would seem that the icebox method searches out the syphilitic process not discoverable by the water bath fixation method, and, in our experience, positive results were never obtained in nonsyphilitic nervous diseases.

DISCUSSION

Dr. David J. Kaliski (by invitation): I have used the icebox method for many years. The facts mentioned by Dr. Stevenson as to the superiority of this method have been brought out by many writers. Incubation in iced water may be even more effective than the dry method. The method is a safe specific one, if not carried on too long. Like other procedures, it has its dangers. If carried on for about eighteen hours or more, there are a definite number of nonspecific reactions. With certain limitations, up to four hours for instance, the method should be used as a routine procedure in all Wassermann test laboratories. There is a possible explanation for the large number of negative results reported by Dr. Stevenson, in cases of tabes, although my experience does not confirm this point. When we performed only water bath incubations, our percentage of negatives was not so low in a large series of cases. One has to take into consideration the amount of natural hemolysin present in different serums. Unless that is considered, the weak reactions would not be detected. In the later stages of tabes, also, a large percentage of negative reactions is obtained with any method. One other statement should be questioned, that is that a negative Wassermann test is looked on as a sign of cure. We all know that a clinician with experience in nervous diseases does not rely on a negative Wassermann reaction in the blood alone as an indication of a serologic or clinical cure.

Dr. Lewis Stevenson: The technic used in our cases included fixation for four hours, at zero. Negative results were taken as such. Positive results were checked again by the water bath method, and both results reported. The method might be of use in detecting atypical forms of syphilis of the nervous system.

ARTIFICIAL NERVE BRANCHES FOR INNERVATION OF PARALYZED MUSCLES. Dr. Byron Stookey.

By growing artificial nerve branches, I have attempted to solve the problem of innervation of paralyzed muscle when its nerve supply is destroyed so that neither direct nerve implantation nor nerve suture are available. Experimentally, I found it possible to make a nerve grow an artificial nerve branch at any desired level by using a free nerve transplant and direct implantation of the latter into the muscle. Branches were grown from nerves at levels where normally branches are not found—such as from the ulnar and median nerves in the arm and to muscles normally not innervated by the nerve selected. After depriving the biceps of its nerve supply, an artificial nerve branch was grown from the ulnar to the biceps. At various intervals, from 120 to 239 days after the formation of an artificial nerve branch, electric stimulation gave a rapid, full contraction of the biceps. The biceps muscle in each was normal in size and color, showing no fibrillations or the yellowish color characteristic of degenerate muscle. Histologic studies revealed normal muscle fasciculi of normal size and with distinct striations. In both longitudinal and cross sections numerous nerve bundles and nerve fibers were found, thus showing that regeneration and innervation had taken place. Tracings and microscopic sections were

shown. The formation of artificial nerve branches had not caused any apparent paralysis in the peripheral ulnar musculature. However, I believe that in the formation of artificial nerve branches some paralysis may result but that this may be minimized by delicate technic and by selecting certain points on the nerve trunk at which to form the branches. I suggest further that the method may be of value in individual muscle paralysis of anterior poliomyelitis, such as the deltoid, the biceps, etc.

DISCUSSION

DR. I. ABRAHAMSON: Was the work controlled by electrical examinations and what information was obtained from such tests?

DR. OLIVER STRONG (by invitation): I have examined some of Dr. Stookey's sections and have seen the bundles of fibers of which he speaks. It is difficult to demonstrate individual nerve fibers in tissues, although nerve bundles can usually be distinguished more easily. In overcoming this difficulty, modern neurosilver stains are of great advantage. Of course, one might use a myelin sheath stain, but where the regenerating nerve fibers have not reached their full state of development they may not have acquired the sheath, and, in any case, the sheath is absent near the termination of the fiber, so that the choice of the silver stain for axis cylinder staining was wise. In this way, one can see individual fibers here and there in the muscles. Those who are not familiar with the histologic side of the work are likely to suppose that we can demonstrate the nerve end plates in this manner, but this is not necessarily the case, as this stain does not always demonstrate the motor plates even when the fibers are stained up to the termination of the latter.

The criticism might be made that these were sensory nerves and not motor fibers. Aside from the improbability that only one type would be regenerated, the answer to this is that the muscle fibers were in an absolutely normal condition, which, of course, would not be the case unless the muscles were receiving the proper innervation.

It is hard to appreciate, even when hearing this presentation, the enormous amount of work that has been given to the histologic side in regard to the regeneration of nerve fibers. This question of nerve regeneration is important, and there are two principal theories as to the way in which it takes place. One of these theories is that of centrogenesis, according to which the regenerating fibers are outgrowths of the central stumps; and the other is that of autogenesis, according to which the nerve regenerates in situ. The neurolemma cells of the distal stump proliferate and form what are termed band fibers. The autogenists believe that the new nerve fibers are formed out of the substance of the band fibers.

By means of the silver staining methods the axis cylinders, especially the regenerating ones, can be clearly demonstrated. The clearness of the results obtained by this method is shown in the picture of the cross sections of regenerating nerve fibers, which by no means exaggerates the amount of detail which can be made out and the clearness of the preparation. The application of this histologic method shows that the new fibers are outgrowths from the central stump. The band fibers which are formed by the old neurolemma sheaths are important in establishing the old connection with the central nerve fibers, but the regenerating fibers are not formed out of them but grow along or in them from the central stumps to their appropriate destinations. The sprouting nerve fibers, for example, traverse blood clots or enter muscle tis-

sue in which there are no band fibers. The fibers entering the scar tend to branch and thus find their way across the scar to the band fibers on the other side. By following the band fibers they are found finally to reach the vicinity of their former terminations in the muscle fibers. When they reach the vicinity of the old motor end plate possibly some chemotactic influence of the remnants of the old motor end plate attracts the motor fibers to their proper terminations in them.

Some interesting work has been done by Jello and others on the innervation of the motor end plates in the embryo. From this it would appear that they reach the embryonic muscle fibers and come in contact with them before the sarcolemma is formed. This explains the trypolemmal location of the end arborizations and possibly the profound influence of the nerve fiber on the muscle fiber. There is evidently some relation between the nerve fiber and the nucleus of the embryonic muscle fiber. The nuclei of the embryonic muscle cells are central, but later in the course of development they become superficially placed. The growing nerve fibers in the vicinity of a muscle nucleus show thickening. They then arborize and as they arborize the muscle nuclei in their proximity proliferate, thereby forming multinucleated "sole plates."

In regard to the practical application of these latter observations, possibly accelerating chemical substances might be used to aid regeneration. Chemotactic influences probably form an important part of the rôle played by the band fibers and also by the pieces of resected nerves used in Dr. Stookey's experiments.

DR. LOUIS CASAMAJOR: This work is a combination of nerve graft and nerve implantation. The muscle in these cases had had no chance to degenerate. Such a formation of branch is possible, but further work is necessary to show whether a similar success can be obtained in a muscle which has already undergone atrophy. In our operations, we usually encounter muscle which has already become atrophic. These particular experiments should not be applied to the human subject, because we should not care to sacrifice part of the ulnar to get a good biceps. A man could get along with a poor biceps, but he could not manage without a good hand. In the paralyses of poliomyelitis, this method would have a definite application. Muscle transplants have been fairly successful, but the results have not been fortunate. We might obtain neuronization of the muscles, and a great deal might be added to the happiness of the individual. Part of the posterior tibial could be sacrificed, which would be quite justifiable if this could be done after the muscle had degenerated. There might be some return of function, which is the greatest hope that can be held out in these cases.

DR. ALFRED S. TAYLOR: I remember the work done on nerve transplant by Dr. Globus six or eight years ago. He took a segment from a good nerve and implanted it into muscle; the work was not so interesting as this, nevertheless it gave good results. The muscle gave good contractions when the nerve was stimulated. Good end plates were seen clearly in slides from the muscle. I think Dr. Stookey would find them in his muscle if he searched long enough. With regard to dividing a small segment of nerve and not finding paralysis, an Englishman, working with the hypoglossal anastomosis, found that one third of the hypoglossal nerve could be divided without causing paralysis. Other investigators have shown that the fibers of a nerve branch are collected 2 or 3 cm. above its exit from the main trunk, and above that distance the fibers pass freely from one bundle to another. If the nerve trunk is divided

at any point well above a branch, it is conceivable that the nerve damage involves axis cylinders going to any one branch, and the damage is so diffused that peripheral failure occurs.

In regard to poliomyelitis cases, I am rather pessimistic. I performed a number of nerve transplants in such cases twenty years ago. In one or two, there was partial regeneration; in eight or ten flat failure. In the case of a child very well cared for, who had received massage and other treatment for four years after paralysis of the peronei, the neurologist recommended nerve transplantation of the peroneal nerve into the internal popliteal. After six months, there was distinct evidence of regeneration by electric stimulation, but the functional result was never satisfactory. The child could evert the foot slightly, but enough function could never be restored to make it useful. Drop foot persisted, although there was good voluntary movement in the muscles. I would not advise operation in these cases. Poliomyelitis is more or less of a group paralysis and there may be sufficient damage to neighboring nerves to prevent one obtaining a good virile source to tap for a nerve supply when anastomosis is attempted. This is one reason for failure in these cases.

Dr. Charles A. Elsberg: Experiments of this kind in the laboratory will be of assistance to the surgeon. One point I would like to emphasize is that such experiments require an extreme delicacy of technic, which few workers have attained. These fine nerves must be handled gently to avoid damage. Many experiments in the past have failed, or given variable results, because of lack of delicacy in the technic. Another point is that from the gross appearance of the muscle and the muscle contractions there was satisfactory evidence of regeneration even without histologic examination. The muscle deprived of its nerve supply presents a characteristic appearance in the gross. When the muscle is again supplied, its appearance is entirely changed, and it looks just like normal muscle and cannot be distinguished from it. With electrical tests they give the same contraction. The question has been raised as to what would have happened if these muscles had been atrophied. Clinically, we know that old facial paralyses return to normal. Muscles partly atrophied have a hunger for nerve fibers. If Dr. Stookey had been dealing with muscles deprived of nerve supply, the results would have been more striking. There is a good outlook for the application of this work in some muscular paralyses, whether due to poliomyelitis or to other causes.

Dr. M. A. Kraus: No one can question the great importance of these studies from either a theoretical or a practical point of view.

There are two points in this connection which I should like to emphasize. Dr. Stookey is aware of the difficulty of determining the fascicular content of a nerve at a given level. Furthermore, there are many different kinds of fibers at a given point—motor, vasomotor and various sensory types. A distance of 2 cm. might make a good deal of difference in the fascicular content.

The second point is that the work of McKinley of the University of Minnesota, mentioned by Dr. Taylor, must be considered carefully before it is accepted. In my opinion, the conclusions are not all proved by the experiments. Indeed, many of his conclusions seem to contradict his findings. The statement that the fascicular picture on cross-section at various levels of a nerve varies a great deal, is undoubtedly true, but the fibers which make this continual change, in the vast majority of cases, have different functions, such as motor, vasomotor and sensory—not the same function; in other words, there is a continual gathering into a few fasciculi of fibers of motor, sensory and vasomotor

function preparatory to the leaving of these fasciculi as a branch from the main nerve trunk. It follows naturally that there is continual splitting of fasciculi having various functions.

Since McKinley does not note the entire physiologic significance of each fasciculus, his conclusions in regard to motor fasciculi do not bear careful scrutiny. Indeed, some of the fasciculi he describes are those to joints. Second, the experiments in cutting the sciatic and the single experiment using electrical stimulation were performed from 3 to 4 cm. below the sacral plexus between the ischial tuberosity and the great trochanter of the femur, a point so near the plexus that changes in the fascicular topography of motor fibers immediately below would naturally be expected. The peripheral combinations from various segments going to muscles must occur distally to the plexus. I do not believe that the work of Marie, Meige, Gosset, Déjerine and M. and Mme. Mouzon, not to mention that of Dr. Ingham and myself, can be thrown out so lightly.

This all serves to emphasize some of the difficulties of using nerve branches in practical surgery. It does not in the least detract from the great importance of Dr. Stookey's researches in paving the way which will overcome these difficulties.

Dr. Stookey: Replying to Dr. Abrahamson, we did not use any electric stimulation during the progress of the experiments. There is no method, other than that of Lapicque's chronaxie, to determine the downgrowth of the neuraxes; and this method is not feasible in animals. We stimulated the biceps muscle in the operative field during the progress of the studies in two animals. I have no other data concerning the rate of the downgrowth.

Dr. Casamajor properly raised the question as to the wisdom of going into the ulnar nerve to supply the biceps. We used the ulnar nerve and the biceps muscle experimentally, because this seemed to offer the best technical opportunities in the dog. In man, the choice of nerves and the decision to make an artificial nerve branch depends entirely on the paralysis and the results that may be gained. Obviously, it would be unwise to induce a paralysis of the intrinsic muscles of the hand in order to obtain innervation of the biceps muscle. In reply to the question about innervation of old paralyzed muscles, I would refer to the fact that paralyzed muscle regularly regenerates if neuraxes are supplied, providing too long a time has not elapsed and degeneration of the muscle is not too far advanced.

I am aware of the work to which Dr. Taylor called attention. A flap is raised from the nerve and implanted in the muscle. I would like to emphasize the fact that in making such a flap from the main nerve trunk damage to the nerve is many times greater, and the possibility of successful repair is diminished; but with a fine nerve segment, an artificial nerve branch may be made any length desired with infinitely less trauma to the nerve trunk.

Dr. Kraus mentioned Stoffel's work on funicular anatomy. I do not believe there is a definite funicular anatomy any great distance from the point at which the nerve branches arise. The funiculus to the pronator teres is almost a definite nerve within the sheath of the median. Other examples are to be found in the sciatic, but with these exceptions I do not believe a distinct funicular anatomy exists from the origin of the nerve to its peripheral distributions. Langley and Hashimoto have shown that throughout the course of the sciatic nerve, nerve plexuses which rearrange the nerve fibers are numerous. Borchardt and Wjasmenski (*Beitr. z. klin. Chir.*, 1917 and 1919)

have shown by elaborate studies and dissections on especially prepared nerve trunks, using macerated specimens, that there are no definite funicular paths from the origin of the nerve to the periphery, but that there is a constant interlacing and innumerable nerve plexuses. I believe the only funicular anatomy found is immediately above the origin of nerve branches, where there is a shunting of definite neuraxes on the formation of the nerve branch about to be given off. In certain nerves and certain positions, such as the musculospiral in the middle third of the arm, the small funiculi are grouped together into one large bundle. In selecting the points for formation of artificial nerve branches an intimate knowledge of the finer anatomy of the nerves concerned is presumed.

Dr. Taylor mentioned what is apparently anatomic regeneration without functional regeneration. Complete functional regeneration depends on the afferent proprioceptive impulses as well as efferent impulses. I remember a tailor, who, during the war, had a median nerve suture performed. There appeared to be complete regeneration, but the man did not regain function so as to be able to return to work. He could hold his scissors but could not use them accurately, probably due to a loss of the muscle tendon sense; and he had what could be termed a peripheral apraxia.

A CRITICAL REVIEW OF THE THEORIES OF THE PATHOGENESIS OF EPILEPSY, WITH A NEW INTERPRETATION OF THE AVAILABLE DATA. Dr. Michael Osnato.

The various important theories of the pathogenesis of epilepsy were briefly discussed. An attempt was made to construct synthetically a conception that would harmonize all the available data. The chief feature in the conception offered was based on the work concerning the biochemistry of the blood in epilepsy.

Briefly summarized, this work calls attention particularly to the defective metabolism of the starches in epileptic persons. Organic acids, chiefly acetic, butyric, lactic and tartaric acids, are formed during the second stage of the breakdown of starches by the amylolytic ferments. In normal persons, these acids are changed by the action of the liver and the small intestine into salts, and they are then oxidized into ammonium carbonate or sodium carbonate and urea. In epileptic patients, however, these acids are found unoxidized in the urine and the blood.

It has been proved that in an acid medium, the nucleohiston content of cellular nuclei is broken down into nucleinic acid and proteoses (albumoses). These substances cause the convulsions of the epilepsies.

The acids mentioned, especially lactic acid, disturb the vasomotor tone of cerebral blood vessels. In the generalized convulsions, the unconsciousness is caused by the sudden vascular shutdown, when these toxic substances reach the brain.

In the smaller jacksonian type of seizures, the production of these substances (proteoses) may occur locally in the brain tissue or may be imprisoned locally by sharply circumscribed vasoconstriction.

DISCUSSION

Dr. Louis S. Aronson: It is of interest to note that borax as a remedy for epileptic convulsions was put forth by Gowers many years ago. During the recent war, the French had great difficulty in obtaining phenobarbital (luminal,

or gardinal as they call it), and in order to avoid bromid therapy, attempts were made to return to some form of borax for the control of the epileptic seizures. Marie and Bouttier, after trying potassium and also sodium tetraborate, obtained good results with potassium borotartrate. This is not a mixture; it is a chemical which apparently has some direct effect on intestinal chemistry and the fundamental reactions that have been alleged to be the basis of the convulsive attack. The authors of this form of treatment assert that following its use for several months there is in a given number of cases a complete cessation of convulsions and in the great majority of cases a marked diminution in the number and severity of the attacks. Most patients have 50 per cent. fewer attacks, and many of the attacks are converted from major into attacks of petit mal. Marie and Bouttier do not claim any special cure with this mode of treatment, and, in fact, administer a small dose of phenobarbital to many of their patients once daily, when tartroborate of potassium fails them. They claim, however, that this prepartion can replace the bromids and accomplish as good results as the bromids as an adjuvant to phenobarbital, without producing the undesirable acne, indigestion, impoverished memory and weakened sexual function that one so frequently finds in the chronic bromid user. I have seen many of their patients that were free from attacks for nearly two years on borotartrate medication alone, and many that were benefited to a considerable degree and happy that they were not being given bromids. My own experience is limited to the use of the drug for the last four months, since I have succeeded in obtaining some from Paris, and all that I can say for the present is that it seems to control some of the cases. It is an extremely hygroscopic crystalline flake that cannot be prescribed other than in small vials of one-half to two drams (1.37 to 7.5 c.c.), two to three times daily, for the patient to dissolve and drink in fresh solution.

DR. FOSTER KENNEDY: The high degree of carbohydrate fermentation found in the intestine is of much interest. The relation to intoxication is more probable than to nervous instability. The fact of deprivation of the blood supply seems to be the chief factor. I tried on myself the experiment of compressing both carotids, and before I could count two lost consciousness. The great underlying factor is that of vascular instability. The causes are faulty chemical processes. I believe there is only a difference of degree between the ordinary syncopal attack and that of the epileptic convulsion. The phenomena have the same underlying basis, namely, vasomotor disturbance.

DR. S. P. GOODHART: Did Dr. Osnato consider the aura as part of the epileptic seizure?

DR. OSNATO: In reply to Dr. Kennedy, fifteen years ago, Pike tied the internal carotids and the vertebral close to the subclavian, in cats. Asphyxia and a tonic spasm immediately ensued, having all the characteristics of decerebrate rigidity. Artificial respiration continued during the ten minutes that the occlusion was allowed to continue. Immediately, and for a period of four or five days, the cat would have a series of convulsions, which could be brought on even by stroking its fur, jarring the table or making loud noises. This experiment is explained in view of the biochemical studies of Cuneo, by the fact that when cells are shut off from their blood supply, immediate catabolic changes occur. The nucleohiston breaks down into nucleinic acid and proteoses in the presence of the acid medium supplied in this particular case by the asphyxiated blood.

I am quite certain that epilepsy is not a brain disease, that is, that the disease could not be explained by investigation inside the skull alone. I have

carefully studied fifty-seven cases of epilepsy and found that in only three were there any objective neurologic signs. Two of these cases showed sensory motor types of cortical disturbances. They were traumatic cases with craniocerebral injury. The other case was one of so-called idiopathic type and showed a Babinski sign on one side and a sixth nerve involvement on the other.

Although private cases were few in number, I chose them because they were much better material than larger groups of cases from a clinic. Even in good neurologic clinics, patients are examined only once, usually by men whose training neurologically has not been completed. There are also many other sources of error in the examination of clinic patients. However, I have no doubt that if it were possible to examine thousands of cases of idiopathic epilepsy, the numbers showing actual objective neurologic signs would be few. I believe, therefore, that epilepsy is not primarily a disease of the nervous system, but makes itself felt through it in the convulsion and some other episodes.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY*

Regular Monthly Meeting, Nov. 16, 1922

F. H. PACKARD, M.D., President, in the Chair

TWO CASES OF DYSTONIA LENTICULARIS. DR. H. C. SOLOMON:

There is no doubt as to the diagnosis of dystonia lenticularis in the first case. The important point is that the first symptom was a peculiar contracture of the muscles of the left foot. This caused the patient to walk with an unusual limp; but he was able to run in a normal fashion. Therefore the diagnosis of hysteria was made. This condition continued for about three years and then spontaneously disappeared for a period of six months, when it reappeared. From then on progress was rapid in all parts of the body except the head. The patient developed the characteristic symptomatology of dystonia lenticularis, including torsion of the body, hypotonic and hypertonic condition of the muscles, and marked disorder of equilibration, which, however, never led to falling. After a time, a characteristic dromedary gait developed, and later the patient was unable to walk. The second point of interest was a partial remission, so that at the time of presentation the patient, after having been confined to a chair for many months, was again able to walk and go about fairly successfully.

The second patient was a young lad of 17, who about two years previously rather suddenly developed spasmodic torticollis. Six months before the development of this condition, he had been slightly injured in an automobile accident. A few days before the torticollis appeared, he had had two convulsions, which were the only convulsive phenomena that had occurred. Various therapeutic devices had been attempted for a year, including electricity, massage, gymnastics, a plaster cast, a Thomas collar, reeducation, suggestion, psychologic analysis, suggestion under ether, etc. Slight temporary improvement had followed many of these procedures, but on the whole the condition had become progressively worse.

The patient presents a spasm or contracture of the trapezius and sternocleidomastoid muscles of the left side, with occasional relaxation. A consid-

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* Communications from members of the staff of the Psychopathic Hospital, Boston.

erable degree of scoliosis is present. Occasionally the patient is able voluntarily to straighten his neck. At other times, this occurs spontaneously. There has been a considerable degree of myodemia, which is not now present. The biceps and triceps reflexes are very weak. The other reflexes are active. All the muscles are irritable to percussion; otherwise the neurologic and physical examinations are negative.

It is suggested that this is possibly a case of dystonia lenticularis with the onset of symptoms localized largely in the muscles of the neck, but slowly progressing, the patient having already developed a certain degree of torsion. It is realized that it is not possible to make a definite diagnosis of dystonia lenticularis at this time. There is no method of making this diagnosis, except that of the symptomatology. As shown in the first case, it is possible to have symptoms localized in one part of the body continue for a considerable period before a more generalized spreading takes place; a remission of a greater or lesser degree may occur; considering the onset, the disease may well be considered as hysteria.

The most frequent locus of symptoms at the onset is the leg. Dr. E. W. Taylor has presented a case beginning in the arm and a probable case beginning in the neck. It is suggested that this is a case of dystonia lenticularis with the early symptoms affecting the trapezius and sternocleidomastoid muscles.

DISCUSSION

Dr. E. W. Taylor: In the first case reported, which I have had an opportunity to observe, there has been improvement in the last two weeks. I might add that the patient is extremely euphoric, and is convinced that he will be entirely well and wishes forthwith to begin work at an art school. In this case, there cannot be the slightest doubt as to the diagnosis. The patient shows, in peculiar degree, the characteristic feature of dystonia, which is a combination of hypertonia and hypotonia, as its name implies. If one notices carefully the way he holds himself, one sees that some of the muscles are in a spastic state, while others are in a state of hypotonia. It is characteristic that he has never fallen in the ten years during which he has been afflicted. There is no Romberg sign; he can stand with his eyes closed without reeling. This is an addition to the literature on this rare disease, which a few years ago numbered not more than thirty or forty cases.

Regarding the second case, I should think the ultimate diagnosis would depend on whether or not the condition spreads from the neck muscles. It appeared to me that probably the patient had a torticollis. The sternocleidomastoid and trapezius muscles were markedly involved. With this symptom alone one would hardly be justified in calling the condition dystonia; but the process has already begun to extend to the muscles of the body, as shown in the distortion of the trunk. I should not regard the condition as hysterical; it is probably dystonia, but up to this time it can hardly be distinguished from a marked case of torticollis. Is it not probable that the movements of torticollis may be due to a lenticular lesion, or at least to a disturbance of function in that part of the brain?

TWO CASES OF PELLAGRA. Dr. S. K. Smith.

I am presenting two cases of pellagra to emphasize the apparent increasing frequence of the condition in the New England States. There are about six cases in Boston at the present time.

The first case is that of a Scotch woman, aged 43, who gave a history of chronic alcoholism. The patient had eaten little meat for a year and probably

had used no milk. There was a history of mental change of three months' duration, together with glossitis, diarrhea, a bilateral dermatitis of the hands and a loss of weight of two months' duration. Examination revealed (1) marked emaciation; (2) negative neurologic symptoms, and (3) typical delirium, with complete disorientation, confabulation, memory changes and hallucinations.

The second patient was a man of 38, who also had a history of chronic alcoholism. His dietary history suggested a poorly balanced diet. For about three months there had been: (1) alternating gains and losses of weight; (2) alternating constipation and diarrhea; and (3) dermatitis, starting as a macular eruption. Examination revealed: (1) marked emaciation; (2) characteristic dermatitis of the hands, feet, elbows, forehead and neck; (3) neurologic findings which showed sluggish pupillary reflexes, absent abdominal and absent right Achilles reflexes; (4) simple mental deterioration, with marked memory change, disorientation, confabulation and hallucinations.

A CASE OF DEPRESSION WITH FEELINGS OF UNREALITY. DR. C. H. MORRIS.

A married woman, aged 26, gave the following account of the day during which she had lost the data of personal identification: "I left my house in the morning to go to church. Everything at home seemed natural. In church everything seemed different. I did not understand what the priest was saying. When I came out of church I wandered around and got on a car. I think I changed cars several times. I did not know where to go. I could not remember where I wanted to go. I could not remember who I was. I sat all day in a field near Randolph. I knew I couldn't stay there, but I did not know who I was or where to go. Then I walked around and then rode on cars some more. I think I was over in Cambridge, and I think it was 8 o'clock when I remembered my telephone number and everything came back. I called up my sister and told her I would come right home. I came out—I intended taking a car, but forgot again. I walked up and down streets and then everything came back and I went right home. I think it was about eleven when I got home."

How are we going to interpret this loss of the data of personal identification? In this episode it appears that the loss was not in the setting of a delirious condition, not in one of disorientation, for the patient was able to take cars, to go to different places, and to do this without attracting attention. The relation of the loss of personal identification to mood is interesting, for though in many cases of depression the patient feels a change in personality, feels that his actions are no longer self-directed, and that the world has lost a familiar quality, this does not lead to an amnesia of name and place. There are other cases besides those of depression in which there is a peculiar alteration in the feeling of personal identity: those cited by Janet and termed psycholeptic crises, in which a patient may have a feeling that he is different, that is, "It is not I who eats, speaks, works. I lack something to give me real existence." Here we have no loss of the data of personal identification, but rather a change in the feeling tone which is at the basis of personal identification. In other cases of amnesia, the patient in a new environment has forgotten all that identifies him with a painful past environment.

None of the types mentioned in the foregoing quite fits the present case: neither a severe depression, depression with feeling of unreality, psycholepsy, nor hysterical type of reaction. The clinical picture is that of a married woman of 26, who had a bereavement in the early part of January, 1922, in the loss

of her third child, at the age of 2 weeks, and who soon afterward developed a pleurisy with effusion. In February, she received a letter from her brother in Colorado warning her of the danger of pleurisy and telling her that it was usually due to tuberculosis. Her brother died of tuberculosis soon after this. In March, she had pneumonia and claims never to have recovered, for she has since had an empty feeling in her head, poor appetite and palpitation. In March, while recuperating, she began reading a medical book with especial reference to tuberculosis and insanity (her mother had died in an insane asylum two weeks after having pneumonia). After reading about hallucinations, she heard a chain dragged across the floor, and knocking on the windows. Since March she had been depressed, had taken little interest in anything, and had been retarded. She entered the hospital in July, presenting at that time a picture of depression and retardation, and did not develop a feeling of unreality until her first visit home in September. On that occasion she said that the home looked "dark and dreary and different from the way it used to." She described her second visit a week later as follows: "I did not know what it was all about in church, but I knew I was in church. I saw all the people and priest, but I didn't know why they were there. At home everything looked queer and mixed up as though the tables and chairs didn't belong there. I could not think." On her third visit home, the incident of the loss of the data of personal identification occurred, and on that same day her two children were found comatose, and one died later at the City Hospital, where a diagnosis of veronal poisoning and bronchopneumonia was made. The children had been alone with the patient on the preceding day. On her return to the hospital she did not remember having been there before, though she had been away five days only, and said: "Everything looks queer. Nothing looks natural. People look as though they were pieces of wood walking around." At first she did not recognize people, and later said that her husband talked about things she used to know about. In this case we have both a feeling of unreality and the loss of the data of personal identification.

In the etiology we have a serious illness from which the patient claims never to have recovered fully, worry over her brother's prognosis in regard to tuberculosis, and worry over the restriction of her family, which seemed necessary on economic grounds and yet which could not be reconciled with the teachings of the Catholic church. The physical status does not show a fully adequate basis for this reaction, nor is this psychosis of the same type as a depression of endogenous origin; this difference of type suggests a different etiology.

DISCUSSION

Dr. F. H. Packard: It would seem to me that this patient was one who simply began to become confused and dazed, and in the beginning events began to be vague. It is a little hard to understand how, at this time, she could remember what happened when she could not remember anything. I should say the explanations she makes at the present time are merely attempts to explain a situation.

PRESENTATION OF FIVE CASES OF MENTAL DISORDER WITH LOW BASAL METABOLISM. Dr. Karl M. Bowman.

Case 1.—An unmarried woman, aged 44, who was admitted to the hospital in a stuporous condition with a history of a cold three weeks before admission, and weakness, became excited and noisy, and finally developed a stupor. The basal metabolism was —56 per cent. As far as known, this is the lowest basal

metabolism on record. Examination revealed a typical case of myxedema. Attention is called to the sparse hair, scanty eyebrows and pubic hair, and lack of axillary hair; the dry, rough, leathery skin with a yellowish tint; the marked edematous and flabby eyelids, and the large, beefy tongue. The urine showed albumin and casts; the phenolphthalein excretion in two hours was 50 per cent. The blood sugar curve was: fasting, 134 mg.; one hour, 144 mg.; two hours, 160 mg.; three hours, 170 mg.

In contrast to the first case in which the low basal metabolism was thought to be due essentially to hypothyroidism, four other cases are presented in which it seems impossible to demonstrate any such cause.

Case 2.—An unmarried girl, aged 22, had been having narcoleptic attacks for the last nine years. The basal metabolism in this case was —18 per cent.

Case 3.—A girl, aged 10, had had typical grand mal and petit mal attacks since she was 10 months of age. The basal metabolism in this case was —24 per cent.

Case 4.—Conversion hysteria was diagnosed in a married woman, aged 40, who for the last year and a half, following some distressing incidents, had developed fainting attacks and typical hysterial anesthesia and paralysis of the right arm. The basal metabolism in this case was —26 per cent.

Case 5.—Schizophrenia was diagnosed in a boy aged 15, who had developed a stuporous condition about a year before, in which he was mute, showed some waxy flexibility, had hallucinations at times, and had to be fed by tube. The condition had followed a blow on the head. The basal metabolism in this case was —20 per cent.

The first case is presented because it is typical of myxedema and probably shows the effect of lack of thyroid secretion on basal metabolism. In the other four cases, the cause of the low basal metabolism is unknown. Careful studies have been made in every case for evidences of endocrine disorder, and the effects of various endocrine feedings are being tried without satisfactory results.

Further studies in basal metabolism at the Boston Psychopathic Hospital have demonstrated a marked tendency to low basal metabolisms in various types of mental disorder. No attempt can be made at present to explain this, and the cases are presented merely to call attention to an interesting clinical observation which may lead to further study, and perhaps give us some further insight into the mechanisms of mental disease.

DISCUSSION

Dr. Percival Bailey: Dr. Bowman says he has no explanation to offer for the low metabolism. I also have no explanation, only an hypothesis. It seems to me unnecessary to look further than the brain for the lesion responsible. There is in the base of the brain—to be exact, in the hypothalamus—a region which exercises control over manifold visceral and chemical activities in the body (metabolism of sugar, proteins, heat regulation, etc.). It seems to me reasonable to seek the explanation of both the mental disturbances and the disturbances of metabolism in these cases in the same pathologic influence on the brain.

THE ACTUAL ENVIRONMENT OF CHILDREN IN A BOSTON DISTRICT. Dr. Marianna Taylor.

A survey has recently been made of a section of Boston by the Mental Hygiene Association from the point of view of the mental health of the children.

Fifty homes of all types were visited—also kindergartens, day nurseries and clinics. The findings were exceedingly significant.

In an unselected series of 190 children, the following conditions were noted:

1. Speech Defects
   - Baby talk 10
   - Stuttering 1
   - Late in talking 4
   - Mutism 2
2. Motor Habits, Incoordination, and Convulsive Manifestations
   - Fidgetiness and choreiform movements 13
   - Twitching of eyes and face 7
   - Shivering 2
3. Incontinence, etc.
   - Bed wetting 49
   - Incontinence by day 27
   - Soiling 9
   - Difficult micturition 1
4. Conduct Defects
   - Holding of breath 1
   - Tantrums 52
   - Spoiled child (stubborn, difficult, uncontrolled) 41
   - Excessive boldness 11
   - Excessive timidity and shyness 13
   - Excessive finnickiness and precision 1
   - Runaways 3
   - Truancy 2
   - Pilfering 1
   - Biting and scratching others 5
   - Seclusiveness 6
5. Sleep Disturbances
   - Restless sleep 12
   - Disturbing dreams 7
   - Sleep walking 1
   - Crying out in sleep 6
   - Sighing in sleep 2
   - Talking in sleep 11
6. Mental Habits and Mental Grades
   - Mentally retarded or defective 13
   - Children in one grade two years 18
   - Day dreaming and fantasying 3
   - Preoccupation and absent-mindedness 4
   - Epileptic children 3
   - Idiocy 4
   - Mentally deficient, epileptic, or psychotic parents 18
7. Miscellaneous
   - Capricious appetite 17
   - Headaches 2
   - Imitativeness (excessive) 1
   - Masturbation 12
   - Thumb and finger sucking 10
   - Nailbiting 33
   - Picking of nose, lips and fingers 26
   - Gritting of teeth 3
   - Strabismus 10
   - Excessive jealousy 2
   - Hydrocephalus 2

The actual environmental factors and neurotic traits found can best be appreciated by citing a few illustrative cases:

Case 1.—In the home of an Italian laborer the six children, ranging in age from 10 years to 10 months, were found to have all been slow in developing, none talked before 2½ years of age, one is retarded at school, three have enuresis, all masturbate. The 5 year old child set herself on fire recently and was seriously burned; the mother became maniacal and died within two weeks in an asylum.

Case 2.—In an American family consisting of father, mother and three children, the father had recently been a patient in the Psychopathic Hospital for eighteen months. He had a violent, uncontrolled temper, and his wife refused to have him placed in an institution. The oldest child showed many neurotic traits. She was pale and undernourished. She was so morbidly anxious to be on time at school that each night she put on fresh clothes and slept in them in order to be punctual the following morning. The second child had had convulsions several times, and the third child was premature.

Case 3.—In a family consisting of four girls and one boy, the oldest presented a number of neurotic habits; for example, nail-biting, talking in her sleep and picking her nose. She and one sister were retarded at school and had repeated a grade. Two other children had enuresis, day and night; no effort was being made to correct these conditions.

Case 4.—The next family consists of father, mother and eight children ranging in age from 16 years to 5 months. The 15 year old boy had énuresis, the 5 year old girl bit her nails, and the 3 year old child sucked her thumb constantly. She attacked her sister frequently with a butcher-knife. The 16 year old daughter recently ran away and married a man much her senior, because her mother beat her, gave her no freedom and made home unbearable.

Case 5.—The next case is that of an epileptic mother who beat her children and spent the greater part of her time on the streets, leaving her children locked in, in charge of the 5 year old child. One of them was retarded in school, had a speech defect, was a finnicky eater, and was underweight. One slept poorly and had convulsions.

Such findings point to the need for educational and preventive work along the lines of mental hygiene: the establishment of mental hygiene clinics, the instruction of social workers, nurses, and parents, and better recreational facilities and supervision of the juvenile population.

THE CHICAGO NEUROLOGICAL SOCIETY

Nov. 16, 1922

Haim I. Davis, M.D., *Presiding*

AN UNUSUAL SYNDROME: ANOSMIA AND AGENSIA. Dr. H. Douglas Singer.

A medical student 26 years of age, eight weeks ago was thrown from a truck, striking his head in the region of the lambda. The accident happened on a Thursday afternoon, and he remembered nothing before the following Saturday

morning. He had a little headache for a few days, but was able to return to school on October 1, about two weeks after the accident. At that time he had a slight headache, tired easily and noticed that a strong light "bothered his eyes." These symptoms disappeared after a short time. Since the accident he has experienced loss of the senses of smell and taste, at first absolute. Taste is beginning to return slightly, and he can distinguish food which is salty, but there is no sense of smell. He constantly experiences an unpleasant odor, which he compares with that of xylol.

Roentgenograms of the skull show a starred fracture at the point of impact in the apex of the occipital bone just behind the union with the parietal bones and a broad fissure extending forward as far as the coronal suture, completely separating the two parietal bones. On both sides, there is also, for a short distance, some separation of the fronto-parietal sutures. The interesting question is as to the seat of the lesion causing this loss of taste and smell without other symptoms. The combination suggests that it must be cortical and, of course, bilateral. The visual fields are normal; fifth nerve sensibility in nasal and buccal cavities is intact, and there is absolutely no other evidence of damage. There is no history of hemorrhage from the nose or ears.

TWO CASES OF BRAIN TUMOR WITH VENTRICULOGRAPHY. Drs. Peter Bassoe and Carl B. Davis.

This article is published in full on page 178 of this issue.

DISCUSSION

Dr. T. D. Allen: In the first case the muscle findings were not very satisfactory, and the atrophy suggests that there was some pressure irritation or even pressure obstruction of the nerves. It was interesting to find a segmental defect in one eye of this boy. I did not take muscle measurements in the second case. So far as I recall, there was no evidence of paralysis of any muscle, but one would expect from the section that there should be paralysis of some ocular muscle, just as there seems to be pressure atrophy of the second nerves.

Dr. George L. Davenport: I should like to ask one or two questions: First, was the ventriculography performed under local or general anesthesia?

Dr. Peter Bassoe: General.

Dr. George L. Davenport: Were there any brain findings postmortem, to which death might be ascribed?

Dr. H. A. Oberhelman: None except those mentioned by Dr. Bassoe—increased intracranial pressure.

Dr. Davenport: My experience embraces over twenty cases, and in a few of these the injection of air has been repeated. Local anesthesia is used in all, unless the patient is extremely nervous and will not cooperate; then general anesthesia is resorted to. The pictures are taken soon after the injection of air, and it is hard to get clear ventriculograms, owing to the movement of the head caused by the forced respiratory movements when the patient is under, or coming out from the influence of, a general anesthetic.

I have not had a death that can be ascribed to the ventriculography procedure. I might qualify that statement by saying that one patient died suddenly eighteen hours after the operation. Dr. LeCount, at the necropsy, found no

reason for assigning death to the ventriculography procedure. There was a large cerebellopontile angle tumor and we felt that the sudden death of a patient with a tumor of this size and in this particular location was not at all unusual, even when operative procedure is not used.

Possible causes of death from ventriculography are: hemorrhage into the ventricles, air embolism, sudden reduction or increase of intracranial pressure, rupture of a vessel, or the sudden displacement of some vital area.

In regard to Dr. Bassoe's cases, I believe the imperfect results can be ascribed to faulty technic, the ventricles not being satisfactorily emptied and refilled with air. All my work has been done through Kocher's point, entering the ventricle at the junction of the anterior horn with the body of the lateral ventricle. The patient lies with the face down, the neck flexed and head inclined to the side where the needle enters, and the replacement with air is done slowly. The roentgen-ray technic is as important and should be as painstaking as the operative procedure. The essential features are the position of the head on the plate and the angle of projection of the central ray.

As to the interpretation of Dr. Bassoe's pictures, I would say that the first case should have shown dilated ventricles, and the second an absolute cutting off of the ventricle at its center or in the region of the descending horn, the anterior and posterior horns showing up. In large tumors there are secondary manifestations resulting from edema, and this must also be considered in the interpretation of ventriculograms.

I believe, as Dr. Bassoe does, that the death of the second patient was due to acute increased intracranial pressure; and if this diagnosis could have been made early enough, the pressure might have been reduced by putting in a needle and allowing some of the air to escape, with the result that the patient's life might have been saved.

In regard to the indications for ventriculography: It can be utilized when tumors cannot be localized; in cases in which the patient has been operated on and the tumor not found; in cases of tumor in which the secondary symptoms are so pronounced as to overshadow completely the early localizing symptoms and signs. With our present knowledge of ventriculography, a careful clinical history and examination of the patient are absolutely essential. Ventriculography should be used only as a corroborative procedure, when necessary, or as an aid when it is impossible to obtain a reliable history or satisfactory examination of the patient. Ventriculography is thus in the same position as all other laboratory methods of diagnosis. I want to emphasize my opinion that routine ventriculography should not be practiced.

I am interested at present in determining how small a tumor may be and how favorably it must be located to be revealed by ventriculograms. In a young boy, a calcified tumor about 1¼ inches (3.17 cm.) in diameter, located in the frontal lobe, produced a definite displacement backward of the anterior horn of the ventricle visible in the ventriculogram.

The interpretation of ventriculograms in practice depends on a careful comparison of the ventricles of the two sides. Again, if ventriculography is negative in a case of suspected brain tumor, it should be repeated after a given time if the symptoms continue, just as is done when roentgenograms are made of other parts of the body. One such procedure does not negative the evidence of a tumor growth.

We all agree that any diagnostic method or procedure should be as safe as it is possible to make it. Only by reporting cases and the results observed,

especially cases verified by operative procedure or postmortem findings, shall we be able to arrive at a definite conclusion as to the value of this procedure in its entirety.

Dr. Loyal E. Davis: As one reads the literature on ventriculography and hears reports on this method of brain tumor localization, one is impressed with the total lack of consideration of the normal ventricle. Before any method which purports to recognize a pathologic variation in any organ can be placed in its propery category, the normal appearance of that structure must become well established.

Retzius, many years ago, studied the lateral ventricles by constructing paraffin casts and called attention to a fact which has been lost sight of; that is, that about one third of all brains have a rudimentary posterior or occipital horn. Only recently, I had occasion to study the lateral ventricles in brains which were hardened in situ and then carefully suspended after removal. The ventricles were injected with mercury under fluoroscopic control, and roentgen-ray pictures were taken by Dr. Arthur Metz in an accurately lateral plane, all necessary precautions being taken to obviate errors in technic. Illustrations which I have show that numerous brains have blunted posterior horns instead of the long graceful extremity we are led to believe is normal. The striking variations in the shape and general contour of the body of the ventricle and the angle at which the inferior horn arises can be seen. It is quite obvious that such anatomic variations might lead to serious diagnostic errors. All of the brains so examined were later dissected, and any reason for such variations sought. In twenty-five per cent. of the brains so examined there were marked variations, particularly in the posterior horn.

In thirty-five cases of brain tumor in the service of Dr. Allen B. Kanavel during the last two years, ventriculography has been employed on two occasions. In one case, the right inferior horn was found to be deformed, but before operation quantitative perimetry revealed quadrantic hemianopsia. This fact is a real criticism of our haste in using the method. In the other case, a frontal lobe tumor, the amount of air injected was not sufficient to fill the ventricles. We have observed three rules in using this method: (1) not to change the position of the head after beginning the operation; (2) to withdraw fluid slowly and inject the air more slowly, and (3) to replace air equal in amount to three fourths of the fluid withdrawn.

Dr. George L. Davenport: In regard to the anatomic variations, it is true that the posterior horn presents the greatest amount of variation found. In the pictures of ventricles containing mercury, it appears that the brains had been removed from the skull before the mercury was injected. The occipital lobes rest on the tentorium and thus the posterior horns will be more flattened and pointed. I agree that variations in the size and shape of the ventricles must be taken into consideration and, as stated before, only by comparing the two sides, as in other roentgen-ray work, can a diagnosis be reached.

Dr. Lewis J. Pollock: The brains in Dr. Davis' cases were hardened in situ, an accepted method for the study of lateral ventricles, and roentgenograms were made by Dr. Arthur Metz, whose technic is beyond reproach. I think the findings may be accepted as fairly accurate.

Dr. Peter Bassoe: I am glad that I brought up these two wretched cases, as I think the discussion will serve to clear up the subject. I was particularly glad to hear Dr. Davenport say that a careful neurologic examination should be made first in all cases, and that the air injection should be used only in

selected cases. This helps to counteract the unfortunate, but current, misunderstanding created by Dandy's paper in *The Journal of the American Medical Association* last year, to the effect that all brain tumors can be located by this method, and that a thorough neurologic examination is not necessary. It is also of practical importance to bear in mind the variations in the shape of the ventricles brought out by Dr. Loyal Davis.

CHRONIC TROPHEDEMA (MILROY-MEIGE DISEASE); A REPORT OF TWO CASES. Dr. William H. Holmes.

On June 1, 1892, Dr. W. F. Milroy, Professor of Clinical Medicine and Hygiene in the Omaha Medical College, published an article entitled, "An Undescribed Variety of Hereditary Oedema" (*New York M. J.*, Nov. 5, 1922). It dealt with the case of a clergyman who presented persistent edema of the lower extremities. The family history of this patient showed that in six generations, comprising ninety-seven persons, there were twenty-two cases of this deformity. In five cases, both lower extremities were involved; in sixteen cases, only one lower extremity was affected; but, unfortunately, no mention is made as to whether the edema was on the right or left side. In one case, no mention is made as to whether the edema was unilateral or bilateral. One member of the group had an enlarged foot until maturity, at which time the testicles began to enlarge. As the enlargement of the testicles increased, that of the foot receded, until it was again of normal size. Although this case was not personally observed by Milroy, I mention the point because it is the only reference I have found in which improvement has been reported after the condition had become established.

Some seven years after the observation of Milroy, Henry Meige reported a number of cases and gave to the disease the name of chronic trophedema, by which designation it is still known. In this country, it is occasionally referred to as Milroy's disease, but in continental Europe the priority of Milroy's contribution is frequently overlooked. The term trophedema, without the use of qualifying adjectives, has been employed by Meige to designate all forms of dystrophic edema, the causes of which are unknown, but which appear to be due to some disturbance of the nervous system.

The disease is characterized clinically by edema which is so firm that the area involved does not pit on pressure. The overlying skin is usually glossy white, and may feel slightly cooler than that of other parts. There is no other evidence of trophic disturbance, such as ulcers, bony deformity, etc. The veins and lymphatics of the extremity affected are not visible. Although the disease may result in enormous enlargement of an extremity, joint motion is not restricted. Patients do not complain of pain, heat, etc., but they are constantly conscious of the increased weight of the affected part. Motor, sensory and reflex functions are normal. When the disease is established, it appears to persist throughout life, irrespective of treatment. Meige says that it persists without noticeable prejudice to the general health, but other observers have reported cases in which there seemed to be increased susceptibility to erysipelas infection of the edematous part. The disease may be congenital; sometimes it is congenital and hereditary, as in Milroy's cases; and it is also observed in adolescents whose family history is negative. It occurs more frequently in the female than in the male. In reading case reports, it has seemed to me that the left lower extremity is involved more frequently than the right. The entire extremity is not necessarily edematous, but only a segment. In order of frequency, then, the disease occurs in a segment or an entire lower extremity,

both lower extremities, one upper extremity, both upper extremities, face and thorax. It is frequently confounded with elephantiasis and with myxedema.

Case 1.—Mrs. J. P., a white woman of American parentage, aged 42 years, first noticed an increase in the size of the right lower extremity about twenty years ago. Shortly thereafter a similar enlargement of the left lower extremity was noted. The swelling gradually increased until at the present time the ankles are 18 inches (45.72 cm.) in circumference. The swelling was not associated with or preceded by any subjective sensations other than a disinclination to move about because of the great weight of the limbs. For many years the condition has remained unchanged. She has only one other symptom, namely, great difficulty in staying awake. Her personal history is negative. She does not recall any serious illness, but assumes that she had all the ordinary diseases of infancy. Menstruation began at the age of 13. It has always been irregular, lasts for three or four days, and on the first day she has some pain. She was married at the age of 28, six years after her legs had become enlarged. Although no contraceptive measures have been used, she has never been pregnant. Her normal weight is 140 pounds (63.5 kg.), but recently she has gained about 8 pounds (226.8 gm.). None of the other members of her family are affected.

On physical examination, the negative findings to which I attached importance in arriving at a diagnosis were: a normal red cell count, white cell count and hemoglobin. A negative blood Wassermann reaction was obtained with two different antigens. The urine was normal. Results of examination of the heart and vessels, of the lungs, and of the abdomen and pelvis were negative. There was no adenopathy. The positive findings were: a hard edema of the lower extremities of twenty years' duration; dry, harsh and scanty hair; a small hard nodular thyroid gland; a retroverted infantile type of uterus; an inequality in the size of the pupils (left larger) with otherwise normal eye reflexes; a lack of adequate emotional reaction, with retarded intellectual processes. Neurologic examination was otherwise negative. The patient would not consent to the making of a photographic record, nor would she enter the hospital so that her metabolic rate could be determined. In arriving at a diagnosis, I considered: (1) the possibility of an obstruction of the lymphatic vessels, (2) an obstruction to the venous return, (3) a metabolic disturbance leading to edema. In regard to the first condition, there was no history of a lymphangitis; the lymphatic glands were not enlarged; a previous filarial infection seemed improbable; and further, the skin did not show the characteristic pigmentation, thickening and falling in folds over the ankle, which is seen in long continued lymphatic obstruction. If the enlargement had been due to venous obstruction there would have been cyanosis, coldness of the extremities, varicosities and a discoverable cause. A cardiorenal basis was excluded by the absence of cardiac signs, urinary changes, hepatic enlargement, etc. I, therefore, came to the conclusion that the condition was based on a disturbance of metabolism due to dysfunction of some of the internal secretory glands. The patient has, for more than a year, received various combinations of thyroid, suprarenal, pituitary and ovarian medication, without improvement.

Case 2.—Miss E. D., aged 30, born in the United States of Scandinavian parents, was examined in Wesley Memorial Hospital, through the kindness of Dr. W. E. Schroeder. The family history was negative. Her personal history was negative up to the age of 15 years, at which time swelling of the left foot was accidentally discovered while having shoes fitted. The enlargement of the foot and leg gradually increased and attained its present dimensions

after the lapse of about eighteen months. She had never experienced any pain. There had been no infections or injuries. She had been admitted to the hospital four times. On her first admission, the blood was examined on three successive nights for filaria, but none were found. As no explanation of the edema could be found, a diagnosis of pseudo-elephantiasis was made. Another surgeon, in an attempt to relieve the edema, buried a strand of silk in the subcutaneous tissues on both the inner and outer surfaces of the leg, extending from the inguinal region to the ankle. She was confined to bed for three weeks, with the foot elevated, during which time the edema almost entirely disappeared. Following her discharge from the hospital, the edema returned almost immediately. Although this woman is 30 years old, she has never menstruated. Her face is infantile in appearance and is covered with a fine downy growth of hair. The hair of the scalp is harsh, brittle and scanty. The bodily development is that of a young boy. The breasts are not developed. There is no axillary hair and little pubic hair. She appears stupid and disinterested.

Examination revealed normal eyes, ears, nose, throat, heart, vessels, lungs and abdomen. Rectal examination revealed no masses in the pelvis. The uterus was small. There was no adenopathy. Blood counts and blood Wassermann tests were negative. Nonprotein nitrogen, blood urea and blood creatinin were within normal limits. Her basal metabolic rate was plus 9. The urine was also negative. The left leg appeared to be about twice the size of the right. There was a white, firm, painless edema which did not pit on pressure, extending from the ankle to the inguinal region. The edema was mostly above the ankle, the foot being only slightly edematous. Roentgenograms of the pelvis and extremities showed no changes in the bony structures. Motion, sensation and reflexes were normal.

Measurements in Cm., Circumference	Right	Left	Difference
At the gluteal fold	47.0	53.5	6.5
Mid-thigh	44.5	49.0	4.5
Knee	34.5	45.0	9.5
Calf	29.5	45.0	14.5
Ankle	19.5	26.5	7.0

THE IMPRESSIONS OF NEUROLOGY IN RUSSIA AND GERMANY.

Dr. George B. Hassin.

During my recent stay of three months in Russia and Germany, I had the opportunity of meeting a great many interesting people. Among them were scientists, teachers, laboratory workers, clinicians and even communists, commonly known as bolsheviks. I also had the opportunity of visiting laboratories, hospitals and clinical institutions, as well as private homes of university professors, school teachers and physicians. From numerous conversations and personal observations, I gathered some information as to the political and economic conditions of Russia, as to the condition of the medical sciences in general and that of neurology in particular.

This report will be limited to a brief outline of the conditions in Russia where I spent nine weeks visiting various cities (Petrograd, Moscow, Kiev, Odessa, Nijni Novgorod, Kherson).

The neurologic work, the clinical part of it, is being carried on in Russia as usual. It never ceased. Even during the frightful years of the civil wars

that, following the bolshevik revolution, brought on terrible misery and poverty, the medical men, including neurologists, continued their hospital and clinical work. Because of terrible surroundings, lack of intelligent help, food, light, heat, animals and chemicals, research work suffered to an extreme degree. Yet, in some instances it was done as scientifically and thoroughly as in the best hospitals of this country.

The clinical material in Russian hospitals, especially in the wards and in the university clinics is very rich. The wards are frequently overcrowded. But the patients suffer, not so much from lack of medical skill, attention or nursing, as from need of medicines, food, and in winter, from the terrible cold. In cold weather they try to keep the patients more or less comfortable by piles of rags, old worn out overcoats and pillows, while the physicians make their rounds in overcoats, felt or rubber shoes and of course with their hats on. Even in the month of September, when the weather in Russia was comparatively mild, an attending neurologist was making rounds with his military overcoat on. This became, he remarked, a habit with them, not only in the hospitals, but also in government civil institutions. I was told that owing to the terribly cold winters, the absence of hot water, few habitable houses, overcrowding (whole families had to live in one room), poor sanitation or rather absence of it, lack of soap and warmth, undressing was out of the question. It was therefore impossible to keep one's body clean, and the result was horrible infestation of the hospitals and private dwellings with lice. In short, the hospitals were not only cold, deprived of light and warmth, but also exceedingly filthy. At the time I visited them, the conditions in some cities (Petrograd, Moscow, Kherson), thanks to the mild weather, were much better.

The clinical material, as I said, is very rich and of great variety. Neurosyphilis, postencephalitic states and brain lesions caused by typhus fever are especially abundant. Typhus fever is exceedingly common, owing to the terrible sanitary conditions alluded to. In winter, typhus fever is pandemic, affecting hundreds of thousands of people. For instance, in 1919, 650,000 cases were registered in Russia, of which 55,000 occurred in Petrograd alone, with a population at that time of 700,000. The death rate was about 10 per cent. It is no wonder, therefore, that this disease became a favorite object of study, numerous monographs and magazine articles having been devoted to various phases of this morbid condition, including the nervous system. Dr. Davidovsky of Moscow, in 1920, published a large work on typhus fever, in two volumes. The first volume of 350 pages has forty pages devoted to the nervous system. Though the paper is poor, the book has a number of good photomicrographs and beautifully colored illustrations, while the title page bears the famous slogan of the communists: "Proletarians of all countries be united." Davidovsky's studies pertaining to the nervous system, based on seventy cases, were carried on in the pathologic laboratory of the first state university of Moscow. They are of great scientific value and considered classic by his countrymen. However, there are a number of minor contributions on the same subject which, in my opinion, are not inferior. I would mention those by Anichkoff of Petrograd, Tiesenhausen and Jguenti of Odessa and Piette of Kharkoff. The specimens I had the opportunity to examine show practically the same changes seen in lethargic encephalitis. Except for the so-called nodules so abundant and constant in brains of typhus patients, there is practically no difference between these two morbid conditions, though it is claimed that in typhus the principle seat of the lesions is not so much in the midbrain, which is the case

in lethargic encephalitis, as in the medulla oblongata. More interesting is the statement that in every case of typhus fever the nervous phenomena predominate, however mild the case may be, and that encephalitis is always present.

Equally great are the opportunities in Russia for studying encephalitic changes in pernicious malaria, which is raging in the eastern provinces of the soviets, such as Ufa and Kazan, and also in some southern regions (Caucasus).

Much less favorable were the opportunities for studying epidemic encephalitis and its sequelae, apparently because for a long time the Russian neurologists did not know of the existence of this disease. Thus the medical association of the city of Kharkov, the capital of Ukraine, was taken rather by surprise when at a September meeting in 1920, Professor Zlatogoroff of Petrograd announced that the Germans had discovered a new disease known as sleeping sickness and that Noguchi had discovered the cause of yellow fever. The first intelligent report on epidemic encephalitis was made in April, 1921, by Dr. I. J. Tarassevitz to the society of neurologists and psychiatrists in Moscow. However, even before Zlatogoroff and Tarassevitz made their reports, cases of epidemic encephalitis in Russia were by no means rare; but they used to pass as atypical cases of typhus or typhoid fever.

The numerous cases of postencephalitic states, so-called pallidal and striatal syndromes, which I saw in Petrograd, Moscow and Kherson, invariably gave a typical history of lethargic encephalitis, and there can be no doubt that in addition to the epidemics of typhus fever, pernicious malaria, recurrent fever and typhoid, there was also one of epidemic encephalitis. At the time of my stay in Russia, this disease was well known and understood. One Moscow neurologist was rather proud of having seen seventeen cases. He was so impressed by his experience that he thought he was justified in writing a book on this disease. When I told him that the literature on epidemic encephalitis in western Europe and the United States was superabundant, that we have excellent monographs by Economo, Hall, Tilney and Howe, he replied that this did not matter. Neurology in Russia, he added, was German, and as the German book on encephalitis by Oppenheim and Cassirer was obsolete, a new book was urgently called for, and he was going to supply the demand.

There is no doubt that many books, pamphlets and magazine articles are being published in Russia, not for the sake of promoting science, but for other purposes. I was assured by competent and reliable men that the present rulers of Russia are trying to promote research. The more one publishes the more chances one has for obtaining food rations, and if a scholar has no contributions to his credit, he is doomed to starvation. Others, again, must show their literary activity in order to be promoted to a professorship, directorship of some institution or to a higher medical degree. In short, the scientific spirit is at least somewhat, and probably very much, darkened by purely materialistic aspects.

The splendid neurologic periodicals of Bechterew, Popoff and the so-called Korsakoff journal of neurology are no more. Small sporadic volumes containing articles on neurologic subjects are being published but very irregularly, some good contributions appearing also in larger general medical monthlies. But special neurologic journals such as we have in this country, Germany, France, England and Italy do not exist in Russia. In fact, serious scientific workers in the field of neurology who would be capable of keeping up a high grade periodical abstain from publishing anything, prefering to do something

else to save themselves from starvation. For instance, a celebrated histopathologist held at one time about four government positions on which he had to spend the whole day, as he had to cover great distances on foot. In four years he has not published a line, on account of lack of animals, chemicals, proper temperature in the laboratory and absence of the most necessary facilities. Yet he is interested in many problems which he is quite capable of solving. For instance, he found that glycogen normally present in the lower portion of the spinal cord in birds is greatly increased when they are fed with rice. His specimens show the presence of this substance, not only in the spinal cord, but also in the subarachnoid space, where this catabolic product is discharged or, as the Germans put it, transported. The study of this interesting problem had to be discontinued; for, as he assured me, though he could obtain rice, he has no means with which to buy hens or pigeons. Before the revolution his family was wealthy. Even if animals could be obtained, he said, they would either be stolen or killed. There are many other problems, such as improving on some staining methods much used in neuropathology, which this excellent investigator had to abandon for lack of proper surroundings. In fact, as he justly remarked, how can one methodically and successfully do research work, especially on a large scale, when the laboratories are bitterly cold or damp, when the limbs are almost frozen, swollen or cracked, when one is underfed, physically exhausted and normally depressed? It is quite true that some exceptionally vigorous persons were doing brilliant work even under such frightful conditions. Thus it is related that the famous surgeon Veliaminoff, although literally starving and freezing (he had to hunt for a warm place), succeeded in completing important books, such as "Surgery of the Joints" and "Military Field Surgery"; and at the time of death he was writing a textbook. He died, they say with a pen in his hand. This occurred at the time of the bolshevik reign of terrorism, when the intellectual class was practically doomed.

To the horrors of starvation, cold, insufficient clothing and filthy surroundings should be added absence of foreign medical literature. During the time of blockade, when letters, books and parcels of any kind were being refused to Russia, no literature could reach that unhappy country. Even at the present time, when it is possible to obtain from abroad what some over there call "mental food," the great majority of scientists are deprived of the means to do so. The common cry is: "Give us mental food" in the form of books, reprints, periodicals, pamphlets or monographs. They can offer nothing in exchange, except rich pathologic material which they possess in abundance, but which unfortunately can be utilized very little. Neither can they preserve it for the future, for lack of containers and necessary preservatives.

The priceless material from typhus fever, starvation and various psychoses is either only partially utilized or has to be thrown away. Especially is it to be regretted that brains from persons who have died from starvation had to be destroyed. The starvation problems have been studied clinically, statistically and physiologically; even the general pathology was covered. Three volumes on starvation have been published by the health commissariat of the city of Moscow. A book on so-called epidemic dropsy or "Oedem-Krankheit" of the Germans has been published by Dr. Luria of Kazan. It contains good descriptions of the changes in the muscles and some ductless glands (thyroid, parathyroid and testicles), but nothing on the condition of the nervous system.

This he informed me he did not study because he did not have the necessary facilities. Nor did anybody else study the condition of the central nervous system in starvation, a problem of great interest. Unfortunately, they expect another famine this winter, especially in southern Russia, and there will thus be ample opportunities for such studies.

Starvation brought forth a dreadful psychotic phenomenon—cannibalism. Cases of the latter undoubtedly occurred, not only in the Volga regions, but even in the more civilized southern towns, such as Kherson. Some wretches would be satisfied by eating the flesh of children they previously killed, while others would sell it openly in the form of sausages and cutlets. Some of the former group of cannibals would ultimately commit suicide, while those of the latter variety were often found to be insane. Even more terrible, unbelievable, tales have been related; as, for instance, the using of the flesh of dead bodies for eating, feeding and commercial purposes.

Another strange psychotic phenomenon observed was the complete indifference of the population to the unfortunate sufferers from hunger. Many grown persons, and especially children, were literally dying in the streets, filling the air with moans, groans, cries and pitiful begging for bread. One physician told me it was not possible to concentrate one's mind on any kind of work. Yet one would pass by the dying victims of incredible sufferings paying no attention to them. In six months, there perished from hunger about 13,000 persons in the city of Kherson, which had a normal population of 89,000. In many cases, not a single member of even large families survived, seventy such families having been found dead in one section of that town. The sensorium I was told became so dulled that one was incapable of comprehending or in any way responding to so great a calamity. There undoubtedly are, in present-day Russia, many social and purely scientific problems of great value and interest to a neuropsychiatrist. The entire convulsive catastrophic movement known as the Russian proletarian revolution possesses many traits of a mass psychosis, for which the nervous system of the future generations of Russia will pay dearly. It is a pity that many innocent people and among them excellent men of science had to perish. Some did succeed in escaping death, but living as they do among pathologic surroundings, they are able to utilize only partially the rich material which is of so incalculable value to science. It is also pitiful to witness the gradual though slow decay of the magnificent clinical buildings, veritable palaces, in Petrograd, Moscow and other places, for there are no means with which to keep these places in proper condition.

The impressions are thus exceedingly sad. In fact, the entire state of affairs in the land of the soviets—the industries, common education, higher education, commerce and trade, science, the housing question, etc.—in short, the entire life, is a profound tragedy and misery.

Comparatively better is the situation in Germany. The atmosphere there is not so depressing as in Russia, and though much work is being done in the laboratories and hospitals, it is greatly hampered by the necessity of giving much valuable time to the struggle for existence. On account of the uncertain future, the necessity to think of daily bread, and lack of proper means, the scientists of Germany do not feel happy, they are much depressed, yet they continue in their work; and, like our Russian brethren, they are much to be admired, but still more to be pitied.

NOTES ON NEUROLOGY AND PSYCHIATRY IN AUSTRIA AND GERMANY.

Dr. N. L. Blitzsten.

During my recent stay of five months in Vienna, I was especially interested in the psychanalytic movement which has had a rapid growth during and following the war. At present, there are in Vienna about fifty analysts in the Freudian entourage. About 30 per cent. of these are laymen, and a large percentage of the others are physicians without previous training in neurology and psychiatry. The Psychoanalytic Society of Vienna is composed entirely of Freud and his disciples. At the meetings, which are held about once a month, the dominating influence of Freud is felt at all times. There is seldom, if ever, any sidestepping from Freud's original postulates, merely further enlargement of them. Practically all of the psychanalytic instruction is in the hands of the Freudian group. During the time I was in Vienna, there were no courses in psychanalysis given at the university; and only one psychiatrist, Paul Schilder, was giving occasional psychanalytic demonstrations in the Wagner-Juaregg clinic. The method of instruction is based primarily on individual analysis of the student, and secondarily, on a series of lectures on theory by members of the Freudian group for those students who are being analyzed. In addition, several of the older analysts devote one or two evenings a month to a discussion, for younger analysts, of analytic problems. In Berlin, conditions were similar. Lectures were given in the Psychanalytic Polyclinic by Karl Abraham and Hans Sachs. Here the younger analysts have the opportunity to carry on their analyses under the direction of the older men.

The duration of a Freudian analysis is about six months, although this is decided during the course of analysis and depends on the overcoming of resistance. No cure is promised, and the patient is told that if the analysis ends unfavorably, the analyst is not to be held responsible. A certain degree of sexual abstinence is asked of the patient. No important decision is made during the analysis. The analyst must not show too great an interest in the cure and should never go beyond the limits of therapeutic interest. The important factors in an analysis are the overcoming of the resistance and the handling of the transference, which is in the end destroyed. It is especially important to know when to analyze the transference. Suggestions of any kind should not be made if the transference is to be handled successfully. The chief work is the affect work. In the analysis, the patient lives through all of those incidents which became pathologic in childhood. The analyst becomes the representation of those figures important in the life of the analysant. At the end of the analysis, the affect neurosis becomes a transference neurosis, which, in turn, disappears with the successful analysis of the transference.

In Vienna, as elsewhere in Europe, there is a strong reaction against psychanalysis. This is due not only to the natural hostility of the conservative minded to the extreme tenets of the Freudian school, but also to the fact that an ever increasing number of laymen and physicians untrained in neuropsychiatry are practicing this specialty. Incidentally, I might mention that hypnosis for investigation and therapy is increasing in popularity in many of the larger neuropsychiatric clinics.

Owing to economic conditions resulting from the war, the progress of neuropathologic research has been greatly retarded. Valiant efforts to maintain previous high standards in this field have been rendered almost futile in some instances because of the lack of funds for even ordinary equipment.

Few are able to devote much time to research because of economic pressure. Many have assumed an attitude of extreme hopelessness because of chaotic conditions.

Clinical neurology has made greater progress, and the clinics of Austria and Germany offer a wealth of material. Especially numerous are cases of neurosyphilis and postencephalitic states. In spite of the ever increasing burden of upkeep, the hospitals are full to overflowing.

Especially interesting in the Wagner-Juaregg clinic in Vienna is the treatment of general paresis and tabes by subcutaneous injection of tertian malarial parasites. Many favorable results are reported, especially when the treatment has been given early in the disease. I had occasion to examine a small group of patients shortly before and after the treatment was given, and I was greatly impressed by the marked and rapid improvement. Schacherel, in the same clinic, uses some form of fever therapy in all forms of neurosyphilis, in addition to the usual antisyphilitic measures. He obtains his best results with typhoid and staphylococcic vaccines injected intravenously. Comparatively little intraspinal medication for syphilis is used in most of the clinics of Europe.

Redlich has been experimenting with inhalations of chlorylen (trichlorethylene) in the treatment of trifacial neuralgia. In many cases complete relief of pain is obtained, especially when the malady has existed for a short time and only one branch of the nerve is involved. Several of the men in Vienna are treating myoclonias and parkinsonian syndromes following epidemic encephalitis with intravenous injection of the patient's own spinal fluid, and they are obtaining favorable results in a large proportion of cases.

In Berlin, where I spent four months, Krause and his assistants are doing considerable experimental work on the autonomic nervous system. They have shown that there is a passage of calcium and potassium from the circulating blood in any stimulation or paralysis of the vegetative system. An isolated stimulation of the sympathetic or parasympathetic nerves by toxins which act peripherally is impossible because of the activity of a regulating mechanism located in the subthalamic centers. Assuming that the proportion of potassium and calcium ions is an expression of the functional conditions of the cells innervated by the parasympathetic and sympathetic nerves, the electrolitic balance must change in favor of one or the other ion in vagotonia and sympatheticotonia. Preponderance of potassium corresponds to parasympathetic stimuli; preponderance of calcium, to sympathetic stimuli.

F. H., Loewy is trying to demonstrate a functional relation between the corpus striatum and the liver. In animals whose livers had been damaged by metallic salts, such as manganese, or by hemolytic serums, he found decomposition ferments in the serum against liver and brain, as well as degenerative changes in the corpus striatum.

In the treatment of epilepsy most of the clinics in Germany employ phenobarbital and bromids in combination rather than separately. Peretz believes that many cases of idiopathic epilepsy are of spasmophilic nature. He treats all these patients with intravenous injection of calcium chlorate, in addition to the usual medication, thereby obtaining, he believes, a larger proportion of cures. Peretz also has found a decrease in the lipoid substances in the serum of syphilitic persons, especially lecithin. He believes that many symptoms of tabes and other forms of neurosyphilis are the result of a disturbed function of the nervous system, which is caused by the chemical combination of the syphilitic toxin with the lipoids of the nervous system. By introducing lipoids into the system, this chemical process can be retarded or stopped, and those

symptoms which are not due to actual degeneration of the nervous system altered. On this basis, he increases the lipoid content of the diet of his neurosyphilitic patients in the form of butter, eggs, cod liver oil and lecithin. This, in addition to the usual antisyphilitic treatment, results, he claims, in a much more favorable outcome in all forms of neurosyphilis, especially tabes.

In the field of psychiatry and the psychoneuroses, there is a general tendency throughout Germany and Austria to break away from the old classification of disease units. As a result, a group of phenomenologists has arisen who make intensive psychologic studies of individual symptoms. The tendency is to group psychologic and biologic types that merge into each other and show evidence of unusual biologic and phylogenetic tendencies which are preformed in the psyche and are brought out in the psychosis but not caused by it.

Intelligence testing among psychiatrists has made little headway in either Germany or Austria. There are no standard performance tests or general psychometric scales in use by psychiatrists. The mental status is diagnosed by individual judgment based on personal clinical experience. The work in mental tests by German psychologists apparently has not penetrated the domain of child psychiatry. The work of America in this, as in other fields, is practically unknown in Germany. This may be due to the prohibitive rates of American periodicals at the present exchange.

The clinics of Germany and Austria still have a wealth of material, and although few Americans are taking advantage of the opportunity, they still attract a large body of other foreign students. Despite the general anti-American feeling throughout Germany and Austria, I saw no evidence of hostility toward American physicians in any of the clinics which I visited.

PHILADELPHIA NEUROLOGICAL SOCIETY

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C. H. FRAZIER, M.D., *President*

A CASE OF SYPHILITIC HEMIPLEGIA WITH UNUSUAL TROPHIC DISTURBANCES. DR. C. A. PATTEN.

A case of hemiplegia was presented with trophic disturbances, consisting of marked destruction of bone and of bedsores, limited to the paralyzed side. There were probably two factors responsible for this condition: (1) the intense syphilitic disease, and (2) the sensory loss.

A colored man, over 60 years of age, was admitted to the Philadelphia General Hospital, July 28, 1922. He had had a stroke five years before, from which he recovered; a second one occurred eight months before and was followed by a semiconscious state for two weeks.

The clinical findings on admission were: the usual manifestations of right hemiplegia with involvement of the lower half of the right side of the face; marked disturbance of all forms of sensation on the entire right side of the body, including the sensory division of the fifth cranial nerve; decubiti on the shoulder, sacrum, hip, ankle and leg of the right side of the body; marked edema of the right upper and lower extremities. Incontinence of urine had persisted since admission. On the left side, the reflexes were prompt and sensation was normal. Argyll Robertson pupils, tremors of the face and left arm, motor

aphasia and mental deterioration were also present. The blood and spinal fluid gave strongly positive reactions for syphilis in all tests. Roentgen-ray examination revealed the following: "Inner surface of the head of the humerus on the right has a ragged outline, the posterior border of the glenoid cavity is not demonstrable, with bony proliferation about this area. Roughening of the outer border of the external condyle of the right elbow, bony proliferation of the great trochanter on the right, and slight erosion of the outer condyle of the femur are present. There is a suggestion of thickened periosteum of the inner upper one-third of the tibia. No pathology of any of the bony structures of the left side of the body or of the skull can be demonstrated."

Under proper care the decubiti began to improve, but the improvement was more rapid under small doses of neo-arsphenamin. At the present time there is only one unhealed lesion, which is on the leg. The incontinence has decreased, and sensation is less involved on the right half of the body.

The hemiplegia is undoubtedly on a syphilitic basis, and the sensory loss on the right side of the body predisposes to decubiti. The presence of an intense systemic syphilitic infection, the loss of mobility of the right side and sensory changes probably account for the marked bony changes.

DISCUSSION

Dr. Alfred Gordon: In the psychiatric department of the Philadelphia General Hospital at the present time is a patient who has shown some mental phenomena suggestive of paranoia and whose history is exceedingly interesting from the point which Dr. Patten has introduced. Before he entered the Philadelphia Hospital, the patient had been in another institution for cerebrospinal syphilis. He presented a deplorable condition—involvement of the sphincters, eyes and upper extremities. The condition progressed, and he was unable to move any of his limbs. He began to develop progressive muscular atrophy of the Aran-Duchenne type, later with involvement of the muscles of the shoulders. Energetic doses of neo-arsphenamin brought about improvement, and the patient was able to walk, the muscles of the hand began to fill out, and at the end of eight or nine months the condition of the sphincters improved. I saw him at the Philadelphia Hospital recently; he was able to walk, had full use of his limbs, and had no disturbance of the sphincters. The most remarkable feature was the disappearance of the muscular atrophy.

Dr. Charles K. Mills: This case is interesting in connection with the question of the possible existence of special trophic centers in the cerebral cortex. We are all familiar with many cases of hemiplegia in which there is little atrophy, although there may seem to be wasting from disuse, and we are also familiar with many cases of hemianesthesia of cerebral origin in which little or no trophic changes are present in bone or muscle, or even in the skin or subcutaneous tissues. Many years ago, Savill and another English medical writer reported cases which they believed indicated that there were trophic centers independent of sensory and motor centers proper. I have seen some remarkable cases of trophic disorder in hemiplegic cases, one in particular in which extraordinary atrophy was evident on the side of the hemiplegia.

A CASE OF PARAMYOTONIA. Dr. Alfred Gordon.

The classical myotonia of which Thomsen's disease is an example presents some variations as to the onset, distribution, etiology and mechanical irritability. The chief characteristic symptoms are: sudden rigidity of the muscles

on attempt to use them, increased mechanical irritability and finally special electrical reactions. A galvanic current will produce a wavelike contraction of the muscles running from the cathode to the anode, also gradual relaxation. Each of these characteristics may vary in intensity. Eulenberg and others described a variety called paromyotonia, in which the muscular spasm occurs not on motion but on exposure to cold, and in which the electrical reaction with Erb's formula may be entirely absent. In other varieties of myotonia there has been a different distribution of the muscular spasms. Myotonic contractions of the muscles of the face have been mentioned by writers, but always in association with involvement of the body. The following case is an example of paramyotonia in which the spasm is confined exclusively to the muscles surrounding the orbits and especially to the eyelids:

A man, aged 52 years, after prolonged mental distress of a domestic character, developed a myotonic contraction of the muscles surrounding the base of the nose and of the orbicularis palpebrarum. Suddenly, without apparent cause, but often on the appearance of a bright light, the muscles around the orbits would contract intensely, the eyes would close tightly, and he would be unable to open them. The condition would persist for many minutes, and relaxation of the muscles would take place rapidly but not suddenly. The patient learned to shorten an attack by a certain turn of his head or by blowing his nose. Further examination, together with the past and family histories, revealed nothing abnormal. Urinalysis and the Wassermann test of the blood were negative.

After a period of treatment with intensive doses of strychnin, the spasm of all other muscles disappeared, but the tonic contractions of the eyelids remained. Mechanical irritability was increased. Testing with electrical currents, galvanic and faradic, gave a prompt response, but at no time was there the classical myotonic reaction of Erb.

The interesting points in this case are: absence of myotonic reactions and of any special etiologic factor immediately preceding an attack; the limitation of the spasm to the muscles of the eyelids which makes this case unique, since in the records of the literature the facial muscles were invariably involved, together with other muscles of the body.

DISCUSSION

Dr. William G. Spiller: In myotonia the contraction is introduced by voluntary movement of the part. I have watched this man, and it is not a voluntary movement that produces the contraction of the lids, nor is the contraction confined to the lids. He has also a spasmodic contraction about the muscles of the mouth. I can see no reason why the condition could not be considered bilateral spasm of the facial nerve distribution.

Dr. Charles K. Mills: I never have seen a case of myotonia like this, if it is myotonia. Dr. Gordon failed to tell us of the refraction and the retinal condition. I have seen cases somewhat like this, which seemed to me to be rather a form of a neurotic or hysterical blepharospasm. It is well known that in certain irritable conditions of the eyes associated with a similar state of the nervous system blepharospasm or a facioblepharospasm is not uncommon.

Dr. Alfred Gordon: I know that in blepharospasm the eyes close and open repeatedly and rapidly, but a condition in which the eyes are tightly closed for as long as ten minutes I do not know. In regard to hysteria, as Drs. Leopold and Mills suggested, the man was examined from every possible standpoint and

that condition was considered. His entire make-up, his previous family and personal history, the absence of hysterical manifestations, not only physical, but mental, absolutely speak against hysteria.

PARALYSIS OF THE UPWARD ASSOCIATED MOVEMENTS OF THE EYEBALLS. Dr. Williams B. Cadwalader.

My object in presenting these cases is to call attention to the occurrence of paralysis of the upward associated movements of the eyeballs from totally different causes. In the first case, there was probably a hemorrhage implicating both oculomotor nuclei, caused by coughing. Similar cases have been presented before this society by Dr. de Schweinitz and Dr. Spiller.

A boy, 14 years old, was admitted to the University Hospital Oct. 25, 1922. In September, 1922, he developed bronchitis with severe attacks of coughing. Soon after an exceptionally severe paroxysm, he noticed diplopia.

On examination, in addition to the signs of general bronchitis, it was found that there was paralysis of the superior rectus muscle on each side. There was decided limitation of the upward associated movements of the eyeballs. There was no involvement of the ciliary muscles. The rest of the examination was negative, except that the patient complained of marked polyuria. This might be attributed to involvement of the tuber cinereum.

The second case was presented here by me and is recorded in the proceedings of the Society (*Arch. Neurol. & Psychiat.* **4**:237 [Aug.] 1920); at that time the diagnosis was obscure because the paralysis of the associated movements was the only striking localizing sign. Since then, however (and it is for this reason that I now call attention to the case), the patient has developed marked disturbance of speech, scanning in character, marked tremor of the right upper and lower limbs, with increased tendon reflexes and ankle clonus on that side, and also intention tremor of the left upper limb. I believe this is a case of multiple sclerosis, implicating the oculomotor nuclei; the symptoms seem to be progressively becoming more pronounced.

DISCUSSION

Dr. William G. Spiller: In the two papers which I have published on this subject (*J. Nerv. & Ment. Dis.*, 1905, and Arbeiten aus dem Neurologischen Institut, Vienna, 1907) I have given all the information I have been able to obtain. In the latter paper, I referred to nineteen necropsies in which a lesion near the aqueduct of Sylvius was found, and to this list I added another case.

I do not believe a lesion far from the oculomotor nucleus is capable of producing a persisting paralysis of upward or downward associated movement, as paralysis of associated movement of cortical origin is always transitory, depending on the bilateral innervation of the nuclei of ocular muscles. The lesion need not be in the corpora quadrigemina, but must be near enough to the oculomotor nuclei to affect the path from each cerebral hemisphere. The lesion may be so small as to escape detection unless a thorough microscopic examination is made. In the case I reported the tumor was so small it might easily have escaped detection. The fact that paralysis of upward or downward movements often is complicated later by paralysis of other ocular muscles also establishes the existence of a lesion near the nuclei.

Dr. J. Hendrie Lloyd: I once saw paralysis of the upward associated movements of the eyes in a case of pseudobulbar palsy. The man had a history of syphilis; he had had several slight hemiplegic attacks, and when he

came under my care he had complete paralysis of the tongue, the lip muscles and of the muscles of mastication and swallowing. Necropsy revealed lesions in the lenticular nuclei. These associated movements of the eyes are undoubtedly represented in the brain cortex; and any lesion that interrupts the connecting fibers between these cortical centers and the nuclei in the midbrain may cause paralysis of associated movements. When this occurs in pseudobulbar palsy, it has been called a pseudo-ophthalmoplegia. The present case is probably not of that kind, but I mention the subject as showing that the lesion in palsy of the associated movements of the eyes is not necessarily in the midbrain nuclei.

Dr. Walter Freeman: It is true that we see paralysis of associated movements of the eyes in such cases of pseudobulbar palsy as Dr. Lloyd mentions, and the differentiation of this condition from the paralysis occurring as a result of interference with the condition below the center is of importance. Bielschowsky has suggested two methods for eliciting reflex movements of the eyes when voluntary movements are no longer possible: one is the use of the aural douche according to the method of Bárány, and the other consists in having the patient fix with his eyes an object directly in front of him, and then turn his head passively to either side. The eyes, fixed on the object, will rotate to the physiologic limit.

Dr. Charles M. Byrnes: Through the kindness of Dr. Spiller, I have just examined the brain of a syphilitic patient who had paralysis of upward associated movement. There was no lesion of the corpora quadrigemina or nucleus of the third nerve, but there was definite infiltration and disintegration of both lenticular nuclei.

SINGULTUS CRISIS IN TABES DORSALIS. Dr. C. M. Byrnes.

A case of singultus crisis in tabes which was perhaps related to a gastric crisis and in all likelihood induced by intradural therapy, is reported.

I could find only three instances in the literature of this symptom associated with tabes. Daunic reported the condition first, and several years later Stembo recorded two instances in which the affection was relieved by induced vomiting.

In my case, the attack developed shortly after the intradural administration of mercurialized serum and persisted continuously for a period of ten or eleven days. The hiccup continued during sleep and while the patient was under ether anesthesia. Various well-recognized methods of treatment were adopted without beneficial effect. Assuming that the singultus in this case was induced by the intradural treatment, it did not seem probable that the mechanism of the attack was dependent on direct phrenic or diaphragmatic irritation, but it did not seem likely that it was due to irritation of the thoracic nerve roots. While it is generally known that the diaphragm receives sensory and motor fibers from the last thoracic nerve, the recent studies of Felix have shown that at least the last six or seven thoracic nerves also enter into the innervation of this muscle.

In two cases of gastric crisis, I have succeeded in relieving the condition at once by means of intradural therapy, and I concluded, therefore, that in this case of singultus crisis it might be well to adopt this method of treatment. Hence, on the eleventh day of the patient's illness an intradural dose of mercurialized serum, containing $\frac{1}{60}$ grain (0.0013 gm.) of mercuric chlorid, was administered. The hiccup ceased immediately, but on the following day there

were several less vigorous and less rapid contractions of the diaphragm. These soon ceased, and the patient has had no further attacks after a period of ten months.

Lantern slides were then shown of the cutaneous anesthetic areas discovered on the thoracic wall, of the diaphragmatic innervation according to Felix and of a diagram constructed by myself to illustrate the intercostal and phrenic innervation of the diaphragm.

NATURE OF CERTAIN FUNCTIONAL NERVOUS DISTURBANCES AND THEIR TREATMENT ALONG METABOLIC LINES. Dr. Ralph Pemberton.

This paper is published in full on page 208 of this issue.

DISCUSSION

Dr. Theodore H. Weisenburg: In a number of arthritic patients who had migraine, improvement in the former disease caused a disappearance of the migraine. This emphasizes the fact that the same underlying causes may exist for arthritis as for symptoms occurring in the psychoneuroses.

In those patients in whom the removal of focal infection does not eliminate the arthritic symptoms, Dr. Pemberton has obtained successful results by limiting the food intake. There is no doubt that a similar method in the treatment of certain types of psychoneurosis will be of value. On the other hand, there are patients in whom overfeeding by the Weir Mitchell method accomplishes the best results.

The treatment of psychoneuroses is by no means a question of the removal of focal infection or underfeeding or overfeeding, but in large part it is a problem of internal medicine in conjunction with the aid of a well conducted biochemical laboratory.

Dr. William G. Spiller: Dr. Pemberton's remarks have historical interest. Charcot some thirty years ago, in his "Leçons du Mardi," spoke of the relation of arthritic disorders to nervous diseases, especially the functional forms; and he emphasized the fact that nervous disorders frequently occur in families subject to arthritic defects. In one place, he says that arthritic manifestations, such as gout, articular rheumatism and cutaneous affections, form a tree; and nervous disorders, such as neurasthenia, hysteria and epilepsy form another tree. The two grow on the same soil and communicate by their roots, and their relation may be so close that one may suspect that they are of the same tree. Dr. Pemberton has given us the common origin in metabolism and biochemistry

Dr. Pemberton: I did not know Charcot had made the statements Dr. Spiller quotes. I knew that the relation had been recognized by many people, but I did not know that Charot had called attention to that syndrome.

Book Reviews

MENTAL DISEASES. A Public Health Problem. By JAMES V. MAY, M.D., Superintendent, Boston State Hospital. With a Preface by THOMAS W. SALMON, M. D., Professor of Psychiatry, Columbia University. Cloth. Price, $5 net. Pp. 544. Boston: Richard G. Badger, 1922.

This is a book full of the statistics and the history of mental diseases. If one prepares to be disappointed on seeing the great number of quotations, one is pleasantly surprised. It is a compilation of the rare right kind.

The first part is developed from Dr. May's work on the Commission on Statistics of the American Psychiatric Association. It emphasizes the importance of mental diseases for every citizen—with an incidence of 566 per 100,000 of the population, an investment of public funds to the amount of $187,000,000, and appropriation of $33,500,000 each year, with a waste from patients out of hospitals which exceeds all these figures. The evolution of the modern psychiatric hospital is traced, with the laws and methods of administration which concern it. Such statements as the following are too often neglected, but will repay the most careful consideration. For the average state hospital reception buildings should be built to care for 6 per cent. of the population, convalescent buildings for 4 per cent., hospital for 2 per cent., infirmaries for 8 per cent., wards for disturbed cases for 20 per cent., for epileptic patients, 3 per cent., planning on quarters for working patients for 40 per cent., the quiet, clean, and appreciative chronic class 14 per cent., and the tuberculous 3 per cent. This and other statements about planning a new hospital are so important that it would be criminal for a commission having such a project in view not to have such sources of information as this book at hand for consultation. Chapters follow on the psychopathic hospital, mental hygiene, immigration, war psychiatry and criminal responsibility. There is a most interesting plea for a better use of the statistical method contained in a chapter on the modern progress of psychiatry.

In a sense, the second division of the book begins in two chapters of Part I on etiology and endocrinology, the former using history and statistics to make its points, and the latter built up entirely of others' opinions in chronologic order. In Part II a chapter is devoted to each of the twenty psychoses listed in the "Classification of the American Psychiatric Association." The method is not that of a textbook, but has an interest of its own. The first chapter, for instance, on "Traumatic Psychoses," refers to ancient writers, then to the crowbar case of 1848, Ericksen's "Railway Injuries" of 1866, the newer ideas brought forward later by Oppenheim and Charcot, and gives in longer abstract Meyer's important contribution in 1903. The extremely interesting "post-traumatic constitution" is well described. The subject is then covered according to Kraepelin and Bonhöffer, with excellent reviews of the character changes which often occur. A few words describe postmortem findings, widespread destruction of nerve cells, glial proliferation and capillary softenings. The chapter concludes typically by quoting the limitations given by the standard classification and a percentage of the admissions of patients with this diagnosis to forty-eight state hospitals (0.3 per cent.).

Other chapters follow the same form, and as the changing ideas that have been published during the centuries and decades are given, a picture of the particular disease is built up which has considerable vividness. There is no attempt to get a cross section of the disease, which would be satisfactory for textbook use. There is a certain looseness in the chapter on alcoholism. We note that in 1909-1912 there was an admission rate of 10 per cent. for alcoholic psychoses to New York State Hospitals, and this rate dropped to 1.9 per cent. in 1920, a prohibition year. When one considers the size of the illicit drug traffic, it is surprising that drug psychoses furnish only 0.39 per cent. of the admissions to state hospitals.

This book is a novel and stimulating one for the psychiatrist! It would be hard to forecast what its effect would be on any one else.

FUNCTIONAL NERVOUS DISORDERS: THEIR CLASSIFICATION AND TREATMENT. By DONALD E. CORE, M.D., M.R.C.P., Honorary Assistant Physician, the Manchester Royal Infirmary. Cloth. Price, $6. Pp. 371, with 21 illustrations. New York: William Wood & Co., 1922.

The purpose of this book is to make a classification of functional disorders which shall be free from the "vagueness" which obtains at present. The foundation for this is sought in certain psychologic concepts, descriptive rather than explanatory, which are outlined dogmatically and in terms which often make reading wearisome. Case studies are conspicuously absent, though illustrative samples are occasionally introduced. Although avowedly dealing with behavior as such, the author insists on the absence of evidence of structural deficiency as a sine qua non for the diagnosis of functional disorder. Apparently, it makes no difference whether the structural defect is such as to determine the behavior in question. Any evidence of feeblemindedness (alluded to as dementia!) or acquired dementia eliminates a diagnosis of functional disorder. Yet one may perhaps legitimately wonder what is the basis for the "defective control of the emotions" on which so much stress is laid. Apparently, "hysterical people, though varying among themselves in their intellectual powers, are as a class less intelligent . . . than those who are not so orientated"; and yet, if the grade of intelligence falls below the imaginary line which delimits normal intelligence, while the person "may show hysterical behavior," this cannot be considered as true hysteria but only as a symptom of the defect.

It is unfortunate that the author has been so ambitious, as there is much in his views that is worthy of serious consideration, but which at present seems to be poorly digested. In dealing with treatment, little use is made of the theoretical considerations which make up the bulk of the book, and the discussions are eminently practical. It seems, however, unfortunate to assert, without proof, that the so-called "mnemoneuroses," which form the "majority of cases of functional nervous disorders as met with in modern civil life," lead almost inevitably, if not remedied, to "absorption of pressor substances, and hyperpiesis—with its consequences, cerebral, cardiac and renal arteriosclerosis."